

**UNITED STATES DISTRICT COURT
SOUTHERN DISTRICT OF OHIO
WESTERN DIVISION**

MELANIE BECKEMEYER,

Plaintiff,

v.

GELCO CORPORATION, *etc.*,

Defendant.

]]
]]
]]
]]
]]
]]
]]
]]
]]
]]

CASE NO. 1:17-cv-00695

JUDGE BARRETT

AFFIDAVIT OF RONALD E. GOTS,
M.D., PH.D., DABT

I, Ronald E. Gots, M.D., Ph.D., DABT, hereby swear, affirm and state as follows:

1. I am over 21 years of age and am competent to execute this affidavit.
2. I am a licensed physician and a Board Certified Toxicologist.
3. As a physician and toxicologist, I have specialized in occupational and environmental medicine and toxicology for over thirty-five years. I have specialized primarily in the determination of cause and effect relationships of injuries and illnesses allegedly arising from chemical, biological (i.e., mold, bacteria and other agents) and other exposures. I have published extensively on the subject of general and specific causation, having written a number of articles and book chapters on this topic. The generally-recognized method of causation assessment in toxicology has been the topic of lectures I have given to physicians, nurses, medical students, attorneys and judges. At Georgetown University School of Medicine, I taught medical students a course on

environmental toxicology in which causation methodology was emphasized. That course included a section on mold, mold toxins and their health effects.

4. I have been involved in hundreds of mold contamination and wet, damp building matters since the late 1980's, including courthouses, schools, other municipal buildings, assisted living facilities, hotels, homes and commercial buildings. I have evaluated health effects, prepared remediation plans, overseen remediation and functioned to ensure that workers or residents not be harmed by any potential exposures. I have reviewed hundreds of scientific papers on mold, mold toxins, bacterial endotoxins, as well as other agents associated with wet, damp buildings, and their surmised, imputed and/or proven health effects and have written numerous articles on the subject. In May 2002, my firm sponsored, along with Georgetown University Medical School, a major international symposium devoted to the health effects of mold and mold toxins. My experience consulting on indoor air quality issues affecting schools, office buildings, residences and even automobiles is extensive.

5. In the past thirty years, as CEO of the International Center for Toxicology and Medicine and founder of Building Health Sciences, my associates and I have been involved extensively in indoor environmental matters. I have examined hundreds of patients, occupants of residential and municipal buildings, as well as schools (both students and teachers) who believed that mold in their facilities was making them ill. I have seen the breadth of complaints, some minority of which may have been mold-related, most of which were not, but were perceived to be so by involved individuals.

6. I have also visited dozens of buildings—schools, homes, apartment buildings, assisted living facilities, hotels, courthouses, and other public and commercial buildings—throughout the United States in which there was water damage, mold growth and concern about

possible health effects. Some of these had exuberant mold growth covering many walls; others, small spots to no observable mold growth. Thus, I have personally seen all of the extremes of water damage and resultant growth of mold. In those investigations, it was often my responsibility to assess health risks, make decisions about removing individuals from the environment and, with my colleagues, to develop remediation plans.

7. Consequently, I have an extensive professional background in the science of health effects from indoor environmental contaminants, the evaluation of individuals who have been in such environments and the investigation of facilities which suffered varying degrees of water intrusions. This, plus my past and ongoing review of thousands of primary research papers, position papers and books dealing with the issues at hand inform my understanding of these matters and my ability to analyze them.

8. I have reviewed thoroughly all of the available records and data in the matter of Beckemeyer vs. Gelco.

9. The basis for my opinions in this case includes my education, training in basic science, experience in toxicology in general and as specifically related to mold spore and mycotoxin exposure, review and analysis of published peer reviewed and accepted literature on the effects of mycotoxins on a broad range of mammalian species including humans, general knowledge of the adverse effects of chemicals on mammalian species including humans, and records reviewed in this case.

10. Mold and mold spores are ubiquitous and the construction and maintenance of a mold-free environment, be it home or vehicle, is not possible.

11. Ms. Beckemeyer believes that exposure to mold and associated mycotoxins in a company car which she drove from June through September 2016 produced a generalized medical

disorder which has led to multiple multisystem symptoms. She is supported in this strange belief by a pediatrician and self-described “mold” expert named Scott McMahon.

12. Ms. Beckemeyer has a variety of complaints, symptoms and diagnoses including respiratory tract symptoms, lightheadedness, vertigo, “brain fog” memory loss, word finding difficulties and anxiety. All of these she and Dr. McMahon ascribe to exposure to mold in the automobile at issue. However, a careful review of the medical records shows clear alternate explanations for these and temporal disconnections-e.g. they preexisted the acquisition of the car or postdated the termination of its use. For example, a major complaint was vertigo (dizziness). However, the first complaint of dizziness bearing any temporal relationship to the car was in October, one month after she turned it in. There is absolutely no mechanism by which such a delayed response could occur. Moreover, she had a long preexisting history of this condition with major endolymph surgery (inner ear operation) for vertigo in 2004, predating the acquisition of the car by 12 years. Many of her symptoms are those associated with anxiety— “brain fog” and sleep disturbance for example. She had a prior and ongoing history of anxiety. She also had changes in her brain seen on MRI which would explain word finding and other cognitive disorders. Those had the appearance of chronic small vessel ischemia seen with ageing and hypertensive disease. She had a long history of poorly-controlled hypertension. Also, molds and mold toxins are not known to produce any brain abnormalities. Her respiratory tract problems during her car usage were diagnosed as ordinary respiratory infections, such as viral sinusitis. Later she was diagnosed as having allergic rhinitis, but she had had that for many years, long antedating the car. Moreover, she was not allergic to any molds associated with the car. Finally, those symptoms persisted long after she no longer had the car—a fact which eliminates an allergic response to the car and indicates responses to other sources of allergens.

13. When the car was tested for wetness and mold, no abnormality was found. There was no source of mold growth and levels in the air and the carpet were low and typical of normal background levels of mold. Every home and every car have levels like those.

14. Several of Ms. Beckemeyer's physicians seem to believe that mycotoxins or other ill-defined "toxins" are responsible for her symptoms. There is no scientific support for this conclusion. One of the claimant's experts, Dr. McMahon, believes that various ill-defined agents in wet-damp indoor spaces produce some sort of toxicological effect which leads to an immunological disorder. He shares that belief with a small cadre of fringe practitioners whose methodology and self-described "disease" is not recognized or generally-accepted. They call this disorder chronic inflammatory response syndrome (CIRS), a "disease" known only to them, not generally-accepted and not recognized by the American Academy of Allergy Asthma and Immunology (AAAI), even though Dr. McMahon and likeminded practitioners claim this is an "immunologic disorder." It has no international disease (ICD-10) classification, the recognized nomenclature by which the US and the rest of the world identify recognized diseases. It is also not recognized by any body of toxicologists even though the claim is that mycotoxins are the initiator of this alleged disorder. Thus, Dr. McMahon's claim that toxins produce this immune system disease which they have personally named CIRS is not accepted by any but their personal adherents. That such a disease exists or that mycotoxins cause this is neither generally accepted by the relevant medical and scientific communities nor is general medical and scientific knowledge. It is an unreliable diagnosis with an inaccurately ascribed causal attribution.

15. An immutable principle of toxicology is that toxicity depends upon the dose of the toxic agent which enters the body. Thus, botulinum toxin is lethal in rather small quantities if eaten by people, but it is injected safely in millions (as Botox) to treat wrinkled skin. By diluting it a

million-fold, Botox is converted from highly toxic to quite safe. The same is true for mycotoxins. The calculated doses required for both acute and chronic exposures to result in adverse health effects in humans have been studied and written about in peer reviewed articles and are so high that it is essentially impossible for exposure in a home, let alone a car, to ever lead to a toxic adverse human health effect. The American College of Occupational and Environmental Medicine (2002 and 2011), the National Academies of Sciences' Institute of Medicine (2004), the American Academy of Asthma, Allergy and Immunology (2006) and the World Health Organization (2009) all concur that the scientific and medical evidence does not support the contention that mycotoxin-related disease (mycotoxicosis) occurs via inhalation of these agents in indoor environments. The current fact sheet of the US CDC concurs with that assessment: <http://www.cdc.gov/mold/stachy.htm> More specifically and pertinent to this case, none of these recognized organizations find any support for the proposition that either mycotoxins or other agents associated with damp indoor spaces, can produce either the range of symptoms alleged here or the "disease" "CIRS" made up by Dr. Ritchie Shoemaker of Maryland and adopted by his protégé, Dr. McMahon.

16. Assuming that the general causation requirements can be met, which is not the case in the Beckemeyer matter, specific causal elements would need to be evaluated and ruled in or out. The causation methodology used to establish specific causation, that is, causation in an individual, is widely-accepted in the literature (Gots 1986, Gots 1993, Hackney 1979, Evans 1976, Irey 1976, Schwartz 1995, Tarcher 1992, Marley 1991, Buffler 1995, Rom 1992, Black 1990, Black 1993, Sullivan 1992, NRC 1992, Brennan 1987, Guezalian 2005, Weiner et al, 2012). Other accepted methodologies differ in detail, but not in basic principle. The elements of the causation methodology used to establish specific causation include the following:

- How was the diagnosis made?
- Does the patient have a recognizable disease?
- Are we dealing with symptoms alone or with objective disorders?
- Have other causes been properly considered and ruled out? Has the exposure been confirmed?
- Was the dosage sufficient considering the concentration and duration to produce the condition(s)?
- Was the clinical pattern what one would expect from that causal agent?
- Were the temporal relationship and/or latency periods appropriate?

All of these questions and their answers are highly individual and must be addressed symptom (or disorder) by symptom (or disorder). Plaintiff's experts, and even some of her treating physicians, have not addressed it in this way.

17. None of the specific causation requirements is met in this case, as the detailed review above demonstrates. In fact, all of them are specifically refuted.

A. How was the diagnosis made? And Does the patient have a recognizable disease?

The primary diagnoses in this case made by standard practitioners were recognized respiratory disorders—allergies and infections. Others were major anxiety disorders. The subsequent diagnoses, and the subject of this claim, CIRS, is not a recognized disease, but the creation of non-traditional practitioners and neither scientifically-known nor generally-accepted.

B. Are we dealing with symptoms alone or with objective disorders?

For some of Ms. Beckemeyer's symptom complaints, specific clinical findings are apparent in the records. These exclusively involve the respiratory tract. She allegedly has certain

laboratory abnormalities, according to several of her non-mainstream practitioners. Those will be discussed later.

C. Have other causes been properly considered and ruled out? Has the exposure been confirmed?

There was no exposure to high levels of mold or mycotoxins in this car. All mold levels were low and/or typical of everyday background levels. There was also no wetness established. Thus, no exposure has been confirmed.

Other causes of her symptoms and findings have been identified including allergies, infections, major anxiety and depressive disorders. She is also clearly persistently anxious which explains many of her symptoms.

D. Was the dosage sufficient considering the concentration and duration to produce the condition(s)?

As a board-certified toxicologist, I can unequivocally state that there were no toxicological agents in this car capable of producing acute or chronic illnesses.

E. Was the clinical pattern what one would expect from that causal agent?

Ms. Beckemeyer's clinical pattern of symptoms, objective findings and response to treatment supported the allergic and infectious conditions diagnosed by her standard practitioners. This pattern is not consistent with the car having been the cause.

That some mold-related or other unspecified "toxin" related to the car was causal of this claimant's made-up "condition," CIRS, is completely unsupportable. This condition has no specific clinical pattern, no objective findings or standard clinical testing; it has a variable array of wide-ranging, subjective symptoms which can occur in virtually any organ system in a pattern defined solely by the patient herself.

F. Were the temporal relationship and/or latency periods appropriate?

The timing of symptoms and the location of their occurrences also speaks clearly against the car as responsible. While Ms. Beckemeyer later claimed that she was sick from the moment she got the car, contemporaneous medical records do not support that. She had acute infectious respiratory conditions for short periods during her three months with the car. She also had allergic symptoms which were ascribed to non-car associated exposures. She later, after disposing of the car, developed most of her symptoms which persisted and, in some instances, intensified. Thus, there is a clear temporal disconnect between the car and her primary complaints.

Very importantly, she had longstanding prior sinus and respiratory disorders as well as major anxiety disorders. Responsible for many of her symptoms, these long predated the acquisition of the car.

The bottom line of this causation analysis is that there is clear evidence against specific causation. The car played no role, except a perceived one, in Ms. Beckemeyer's illnesses or symptoms.

18. The claimant's expert, Dr. McMahon, is relying upon several "evidentiary" approaches to support his causal allegation. All are wrong and defy accepted medical and toxicological reasoning and are therefore no reliable.

A. The first is the general causation belief which I have discussed above. That is, that mycotoxins or other factors coming from indoor environmental air are capable of leading to the manifestations alleged and the strange self-named, unrecognized diseases CIRS. That is untrue, has never been shown scientifically and is not generally-accepted. The US CDC in its current online report agrees (<http://www.cdc.gov/mold/stachy>), as do all of the other consensus groups mentioned above.

B. The second is that the exposure and consequent dose of such toxins allegedly received by Ms. Beckemeyer of mycotoxins or anything else was sufficient to cause the complaints. To “establish” this, the supporting practitioner is relying on several lines of self-developed and self-serving “evidence.” The first is the unproven and likely untrue assertion that “toxins” were present and in sufficient quantities to exert effects. The second, is that those toxins came from the air of the car interior.

C. The third is that the complained of disorders are consistent with toxicity associated with indoor environments.

C. Finally, the fourth is that various odd laboratory tests which he orders, supports his diagnosis and his causal attribution.

19. All of Dr. McMahon’s “evidentiary” approaches as to causation are fallacious, unreliable and misleading.

20. The presence of toxin-producing molds and of mycotoxins or any other “toxic substance” in the car at issue has not been shown. There were no measurements performed during Plaintiff’s period of use, and, the time period between Plaintiff’s last reported use on September 23, 2016 and the testing performed by Plaintiff’s industrial hygienist on July 9, 2018, the car had two subsequent owners. Furthermore, the total mold levels found in the car in July 2018 were not unusually high or indicative of significant water intrusion and active mold growth. They were common everyday exposure levels. Even if the mold spores found were full of mycotoxins, thousands of times more than the largest amount found would have to have entered the body to produce toxicity. As recent studies have shown, airborne mold or mold toxins do not contribute to internal levels of mold toxins. Those come from dietary sources (Follman et al, 2016, Jezak et al, 2016). Nor can it be presumed that those low levels found in 2018 were

sufficiently high at one point in the past and then became low due the passage of time; such logic is not accepted methodology for determining exposure levels. In essence, the ability of a substance to cause a toxic effect cannot be inferred in the absence of information on the dose or to how much of the environmental agent an individual is exposed.

21. If Ms. Beckemeyer were exposed to a mycotoxin, as she and some of her experts seem to believe, and even if they were to enter the body, they are rapidly eliminated. Any mycotoxins theoretically entering her body arising from the car would have been essentially gone by December 2016. However, everyone has levels of mycotoxins in their bodies, arising from dietary sources.

22. Like all other foreign substances, such as medications and most other toxins, mycotoxins are broken down and eliminated by the body. Mycotoxins are metabolized in the liver and excreted by the kidney in short order. Numerous scientific studies have examined the time that it takes for mycotoxins to be eliminated. In toxicology and pharmacology, this rate of elimination is generally called “half-life.” This is the time that is required for one half of the absorbed dose to be eliminated. For the most part, the half-lives of studied mycotoxins occur in minutes to a few hours. Thus, they do not persist for weeks, months or years after exposure cease and, as a result, are not, capable of producing increasing or persistent symptoms or causing new symptoms to arise.

23. Dr. McMahon and a couple of Ms. Beckemeyer’s treating physicians are basically followers of a Maryland physician, Ritchie Shoemaker, who popularized, among a fringe medical group, the diagnoses and testing which they follow. The fact is that these individuals belong to a small cadre of alternative practitioners who have promoted false notions about indoor

environments and all manners of adverse health consequences associated with them. Their beliefs and practices are neither generally-accepted nor scientifically-known.

24. Dr. Shoemaker's practices came under fire by the Maryland Board of Medical Examiners in 2013, when he was reprimanded and put on probation for failing to meet proper standards of care. At that point, he discontinued his clinical practice. However, he now purports to provide special certification to physicians who follow his diagnostic and therapeutic methods. Dr. McMahon, an expert in this case, and a pediatrician by training, touts his mold certification by Shoemaker as his primary credential in this arena. He adds to that the assertion that he has treated thousands of such patients, solidifying his self-proclaimed expertise and diminishing that of anyone who does not have such clinical experience. Medical history tells us that even large numbers of diagnostic and treatment experiences do not necessarily imply knowledge or wisdom. No doubt the colonial physician who bled George Washington would also have trumpeted his experience with thousands of such treatments.

25. Since approximately 2008, Dr. Shoemaker has called his novel disorder Chronic Inflammatory Response Syndrome (CIRS), a condition which is neither generally accepted nor known to exist. Ms. Beckemeyer, and Dr. McMahon use that term or a variation of it, SIRS. Dr. Shoemaker and his followers further claim that this disorder results from exposure to a variety of "biotoxins" associated with wet/damp indoor spaces. This theory is self-generated, unproven, based on poor causation and scientific methodologies, and resides in questionable data. It is not generally-accepted.

26. Dr. Shoemaker originally called the condition Sick Building Syndrome (SBS) and Chronic Biotoxin-Associated Illness, before he renamed it "Chronic Inflammatory Response Syndrome." Initially, he attributed this entity to a mycotoxin cause. In 2002, he consistently

attributed symptoms to “toxin-forming species of fungi” and their mycotoxins. In his book *Mold Warriors* (2005), Dr. Shoemaker wrote similarly about the cause of illness in the cases he discussed. He highlighted water damage and mold growth and that certain molds make toxins, “our studies show repeatedly: mold makes people sick.” (p. 332-333). He has subsequently disavowed the role of mycotoxins, believing instead that, “exposure to mycotoxins is a ‘relatively insignificant factor in the systemic inflammatory response these people get’.” (See Deposition testimony in *Anderson et al vs. The Ritz Carlton Hotel Co. et al.*, DC Superior Court Civil Div. CA No. 05ca (0001130). Now mycotoxins have returned as the alleged culprit as I will note shortly.

27. Certain of the diagnostic tests performed on Ms. Beckemeyer, such as VCS, MSH, TGF, and C4a, even the so-called HLA-genotype allegedly associated with environmental exposure leading to symptoms and a dozen others are neither scientifically-known nor accepted as showing what they are claimed to demonstrate. They are not described in the recognized and accepted medical and scientific literature for this purpose. Many of these tests have little clinical application at all, but are unique to certain specialized research laboratories, or to either specific other disease states or to other illnesses. These tests taken together are not delineated in textbooks or published as part of evidence-based clinical guidelines used by the general medical community. They are the sole province of this fringe medical group who call themselves “environmental physicians,” or, now, “certified mold physicians.”

28. In assessing whether a physician or scientist used reliable scientific methodology to reach his conclusions, it also is important to examine the known or potential rate of error of a theory. For example, if a scientist placed various qualifications on his conclusions, then the known rate of error is potentially very high. The rate of error cannot even be assessed for a

theory that has not been tested. Dr. Shoemaker's and Dr. McMahon's theories remain in the unreplicated, unreliable category. Their alleged scientific methodology in reaching their conclusions about this "CIRS" condition have, therefore, not been confirmed by others or accepted.

29. Another important factor in assessing the scientific reliability of a theory is whether the theory has gained widespread acceptance among scientists in that field. General acceptance indicates that other scientists agree that a theory is based upon reliable scientific methodology, has been replicated and has scientific validity. If, over time, a scientist's opinion has gained little or no support within the relevant field of science and/or medicine, it is appropriate to question whether the opinion is supported by reliable scientific methodology. The term CIRS as a disease entity has been used by Dr. Shoemaker since at least 2008. Dr. McMahon says that the condition was first described in 1997 (McMahon 2017). Since it is, according to them, a widespread immunological disorder (Dr. McMahon now claims 7% of people have CIRS), one would think that if it were proven, by now it would be widely-accepted. It is not. It is not recognized, for example, by the American Association of Allergy Asthma and Immunology, the major organization of immunologists in this country. It also has no ICD-10 number, that is, no number in the International Classification of Diseases, version 10. Dr. McMahon dismisses this, suggesting that such numbers are only for "insurance purposes." Actually, the disease classification numbers are established by the World Health Organization to provide uniform nomenclature for study purposes, so that physicians and scientists worldwide are speaking about the same entity when they do studies. There is no ICD-10 number for CIRS. There is no national or international acceptance of this disease.

30. Dr. McMahon, an expert in this matter, claims proudly to be a disciple of Dr. Ritchie Shoemaker. He diagnoses the Shoemaker disease, now named Chronic Inflammatory Response Syndrome (CIRS), which he believes arises from indoor exposures and attacks every organ in the body. He diagnoses this disease through a set of thirty or more disconnected symptoms and a bizarre array of laboratory tests only performed in this combination by Dr. Shoemaker's zealous followers. This disorder of Dr. Shoemaker's is trumpeted in papers that he has written, most published in obscure, non-mainstream journals or conference proceedings. At this point, approximately eight of his papers have been in the literature for at least ten or more years yet his revolutionary revelation has never found its way into mainstream, peer-reviewed, medical or scientific journals.

31. It has also never made it into any of the most authoritative consensus documents, such as the World Health Organization (WHO) and the Institute of Medicine, National Academy of Sciences (IOM/NAS) publications on wet damp indoor spaces. Although many of Dr. Shoemaker's papers were available by the time the WHO document was published, there are no references within it to Shoemaker or to this "CIRS disease." Dr. McMahon cites extensively from the WHO document and from another, the Government Accountability Office (GAO) document of 2008, which he claims supports him and Dr. Shoemaker. However, neither document mentions any of Dr. Shoemaker's "groundbreaking research," cites any of his papers, or contains any discussion whatsoever of this disease "CIRS." Dr. McMahon's notions, methodologies and diagnosis are neither generally-accepted, nor scientific knowledge, nor reliable.

32. Nowhere in the GAO report which Dr. McMahon cites does it say that we now know of an inflammatory disease described by Dr. Ritchie Shoemaker known as "CIRS" or

“biotoxin illness” or “mold illness” or any other, which produces widespread injury because of a chronically over-reactive innate immune system. In fact, that 61-page document which Dr. McMahon relies upon extensively contains not one mention of CIRS, of him or of Dr. Shoemaker

33. Dr. McMahon claims that this disease, so-called “CIRS,” is an immunological disorder. As noted earlier, it is not recognized by mainstream immunologists or the many thousand members of the American Academy of Allergy Asthma and Immunology (AAAAI), the official AMA recognized body of immunologists. Furthermore, one wonders why, if this disorder is an immune system disease, Dr. McMahon doesn’t refer Ms. Beckemeyer to an immunologist. He, after all, is a pediatrician.

34. A key to Dr. McMahon’s diagnosis of this CIRS is laboratory testing for a variety of parameters which he claims, when taken together, establish the diagnosis. To optimize the so-called “abnormals,” he creates his own normal values which, in some instances, differ from those of the laboratories. When Dr. Shoemaker was challenged years ago about his “normal” value for MSH which differed from the laboratory’s norm or reference range, he asserted that the low end of their reference range was “skewed by all of his patients’ abnormally low values.”. Dr. McMahon has done exactly the same thing in his Beckemeyer report. The fact is that both major commercial laboratories, Quest and LabCorp have an MSH reference range [range of normal values] that start at zero and, in the case of LabCorp, range from 0-40 and, in the case of Quest, range from 0-100. LabCorp is the lab Dr. McMahon used. He insists that the norm should be greater than 35; thus, he asserts, Ms. Beckemeyer was abnormal at 5. In other words, he made up his own normal to fit his own case definition. According to both these laboratories, her MSH was normal.

He did the same thing for the ADH determination which he says was abnormal at Ms Beckemeyer's level of 1. The laboratory which performed the test, LabCorp, reports its normal as 0-4.7. Dr. McMahon says the normal range is 1.3-8 making Ms. Beckemeyer abnormally low, according to him, but normal according to the testing laboratory.

Thus, at least two of these key tests that he relies upon were, according to him, abnormal. His assertion is contradicted by the reference standards of the laboratory which performed those tests and found them normal.

35. In order to avoid evaluation and criticism of his CIRS disorder by toxicologists, Dr. McMahon claims, in his report, that it is not a toxicological disease, but an immunological one. However, in a paper which he wrote in 2017 and references in this report, he says: "In all patients (sic, CIRS patients) environmental exposures to biologically-produced toxins trigger innate immune cytokine overproduction..." In other words, he says in this paper, as he and Dr. Shoemaker have said many times, that their claimed "immune system disease" is the result of a toxic exposure.

36. In an attempt to enhance the respectability and acceptance of his "disease" "CIRS," Dr. McMahon claims that there exist 100,000 references on Systemic Inflammatory Response Syndrome. That disorder (SIRS) is indeed coded in the ICD-9 and ICD-10, as he notes. However, SIRS has nothing to do with his CIRS. SIRS is a code reserved for severe multiorgan-system disease and is used predominantly in patients in ICUs or trauma centers.

37. Dr. McMahon quotes the "Policyholders of America's Research Committee Report on Diagnosis and Treatment of Chronic Inflammatory Response Syndrome caused by exposure to the interior environment of water-damaged buildings (2010)," as the most comprehensive paper on biotoxin-related illness to date. He claims that it shows that the world's

academic community recognizes this disease. It does nothing of the kind. It is a manifesto written by Drs. McMahon and Shoemaker, espousing the same positions promulgated in his report regarding Ms. Beckemeyer. The site "Policyholders of America" is devoted to "mold sufferers" and their supportive physicians. There is nothing academic about it.


38. For all of the above reasons, Dr. McMahon's opinion in this case is far outside the mainstream of medicine. It is not generally-accepted and is scientifically unreliable.

39. Additional and more detailed opinions in this matter can be found in my complete report dated December 1, 2018, attached hereto and incorporated herein as Exhibit 1.

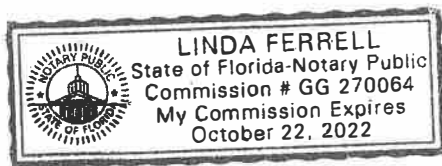
40. My curriculum vitae is attached hereto as Exhibit 2.

41. All of the foregoing opinions are held to a reasonable degree of scientific certainty. This and my report are based on the materials reviewed and analyzed by me to date. Should additional information become available, I reserve the right to amend my opinions accordingly.

FURTHER AFFIANT SAYETH NAUGHT.


RONALD E. GOTS, M.D., Ph.D., DABT

SUBSCRIBED AND SWORN TO before this, the 11 day of April, 2019.




NOTARY PUBLIC



INTERNATIONAL CENTER
FOR TOXICOLOGY AND MEDICINE

CURRICULUM VITAE
Ronald E. Gots, MD, PhD, DABT
Board Certified in Toxicology
Toxicology and Environmental Medicine

International Center for Toxicology and Medicine
93 SW Palm Cove Drive
Palm City, FL 34990-4318
E-mail: regots@ictm.com

Citizenship: USA

EDUCATION:

1961 - 1964	A.B. (Chemistry) University of Pennsylvania Philadelphia, Pennsylvania
1964 - 1968	M.D. University of Pennsylvania School of Medicine Philadelphia, Pennsylvania
1968 - 1969	Internship (Surgery) Johns Hopkins University Hospital Baltimore, Maryland
1969 - 1970	Fellow (General Surgery) Harbor UCLA Medical Center Torrance, California
1970 - 1973	Ph.D. (Pharmacology) University of Southern California School of Medicine Los Angeles, California



PROFESSIONAL EXPERIENCE:

2002 - Present	Chief Executive Officer International Center for Toxicology and Medicine
2003 - 2009	Chief Scientist Building Health Sciences, Inc.
1997 - 2002	Principal International Center for Toxicology and Medicine
1975 - 2009	Medical Director and President National Medical Advisory Service
1995 - 9/96	Medical Director Environmental Sensitivities Research Institute (ESRI)
1984 - 1990	Chairman and Chief Executive Officer Medical Claims Review Services
3/78 - 6/82	Vice President Emergency Medical Services, Inc.
6/77 - 6/82	Vice President Quality Care Management Consultants
10/78 - 1981	Coordinator, Pharmaceutical Class Labeling Project, Food and Drug Administration
1/75 - 9/79	Medical Director and Examining Physician Occupational Health Units, Bureau of Economic Analysis, Census Bureau and Immigration and Naturalization Service, Washington, D.C.
7/75 - 12/75	Emergency and House Physician Northern Virginia Doctor's Hospital
5/75 - 10/75	Medical Officer, Occupational and Emergency Medicine, NASA
1973 - 1975	Senior Investigator/Chief, Department of Gastroenterology, Walter Reed Army Institute of Research, Washington, D.C.



6/73 - 8/73 Emergency Physician
Prince George's County Hospital

1970 - 1973 Emergency Medicine Physician
Westminster Community Hospital
Orange County, California

MEDICAL LICENSURE:

Maryland
Michigan
Pennsylvania
Virginia

CERTIFICATIONS:

1968 National Board of Medical Examiners

1987 Board Certified-American Board of Quality Assurance and
Utilization Review Physicians
Re-certification- 1993, 1996, 1999, 2001, 2003, 2005, 2007,
2009

1994 - Current Fellow, American College of Forensic Examiners International

1996 - Current Diplomate, American Board of Forensic Examiners

2010 Diplomate, American Board of Toxicology

2015 Diplomate, American Board of Toxicology, Recertification

PROFESSIONAL SOCIETIES:

Alpha Omega Alpha (Medical Honor Society)
American Association for the Advancement of Sciences
American Board of Forensic Examiners
American Academy of Clinical Toxicology
American Federation for Clinical Research
American Medical Association
American Medical Peer Review Association
American Public Health Association
American Society of Law and Medicine
American Chemical Society



American Council on Science and Health
Fellow, American College of Forensic Examiners
American College of Occupational and Environmental Medicine
Fellow, American College of Quality Assurance and Utilization Review Physicians
International Society of Exposure Analysis
International Society of Regulatory Toxicology and Pharmacology
National Association of Environmental Professionals
New York Academy of Sciences
Sigma Xi (Scientific Honor Society)
Society for Health and Human Values
Society of Toxicology
Society for Occupational & Environmental Health
Society for Chemical Hazard Communication

TEACHING AND SPECIAL APPOINTMENTS

University of Hawaii - Laboratory Instructor and Tutor, Microbial Genetics. 1964.

Pasteur Institute - Research Fellowship. Paris, France. June 1965 - August 1965.

University of Southern California School of Medicine, Department of Pharmacology -
Instructor, Pharmacology. 1970-1973.

World Health Organization - Temporary Advisor, Workshop on Idiopathic
Environmental Intolerances. Berlin, Germany. 1996.

American Petroleum Institute - Participant, Workshop on Clinical Studies and
Particulate Matter. April 31 through May 1997.

Georgetown University School of Medicine - Lecturer (Environmental Toxicology).
Department of Pharmacology, Division of Toxicology and Applied Pharmacokinetics.
1996 - Present.

Peer Reviewer. *Journal of Medical Toxicology*. 2004-Present

Georgetown University School of Graduate Studies. Adjunct Professor (Regulatory
Toxicology). 2011-Present

University of Virginia School of Public Health - Lecturer, Department of
Environmental Science. 2007



CIVILIAN AWARDS AND HONORS:

1956	Eagle Scout
1967	Alpha Omega Alpha (Medical Honor Society)
1972	Sigma Xi (Scientific Honor Society)
7/71 - 7/73	Special Postdoctoral Training Fellowship, Funded by National Institutes of Health
1973	AMA Physician Achievement Award

MILITARY ASSIGNMENTS:

7/73 - 7/74	Investigator, Department of Gastroenterology, Division of Medicine Walter Reed Army Institute of Research Walter Reed Army Medical Center Washington, D.C. 20012
7/74 - 7/75	Chief, Department of Gastroenterology, Division of Medicine Walter Reed Army Institute of Research Walter Reed Medical Center Washington, D.C. 20012 Honorable Discharge

SELECTED APPOINTMENTS:

National Association of Manufacturers, Medical Consultant, Occupational Disease Compensation
FDA Panelist, Patient Package Insert Hearings
Medical Society of D.C., Member, Workers' Compensation Committee
U. S. Chamber of Commerce, Member, Workers' Compensation Committee
International Association of Industrial Accident Board Commissioners, Member, Medical Committee
Board of Scientific and Policy Advisors, American Council on Science and Health
Chairman, Scientific Advisory Board of NEDA/TIEQ (National Environmental Development Association/Total Indoor Environmental Quality)
Montgomery Medicine, Associate Editor
American Industrial Hygiene Association, Toxigenic Molds Workshop



Hippocrates' Lantern, Contributing Editor
U.S. Congressional Working Group Mold Legislation (2002)
Atlantic Legal Foundation - Scientific Advisory Board
Peer Review, Journal of Clinical Toxicology

SELECTED SPEAKING ENGAGEMENTS AND VISITING PROFESSORSHIPS:

1975	Guest Faculty Member, American College of Physicians.
1980	Invited Speaker, Bendectin Hearings. FDA.
1978 - 1980	Developer and Presenter, Hospital Risk Management Seminars. St. Paul Insurance Company.
1980	Speaker, Annual Meeting. American College of Obstetrics and Gynecology.
1982	Guest Faculty Speaker. American College of Physicians.
1983	Speaker, Annual Meeting. National Legal Center for the Public Interest.
2/85	Visiting Lecturer, Causation and Financial Compensation for Claims of Personal Injury from Toxic Chemical Exposure. International Conference, The Institute for Health Policy Analysis, Georgetown University and the Georgetown School of Law.
1985	Visiting Professor, Legal Medicine. Uniformed University of the Health Sciences.
7/86	Moderator, Medical Section. National Symposium on Workers' Compensation, 10th Annual Meeting, Orono, Maine
8/86	Speaker, "Medical Causation Testimony: Misleading Fact Finders." Workers' Compensation and Employer's Liability Committee, American Bar Association, New York.
1987	Speaker, Annual Meeting. IAIABC.
1987	Speaker, Toxic Tort Seminar. American Bar Association, Chicago, Illinois.



1987	Speaker, Forum Meeting. University of Arkansas for Medical Sciences.
1987	Lecture. Delaware Occupational Medicine Association.
5/88	Speaker, Toxic and Environmental Tort Litigation Committee. American Bar Association, Natural Resource Section.
6/88	Speaker, "Scientific and Legal Approaches to Proof of Causation in Tort Cases." American Chemical Society, Chemistry and the Law Division, Toronto, Canada.
12/88	Speaker, "Medical Cost Containment: Regulatory and Administrative Options for Containing Medical Costs." Workers Compensation Commissioners' Symposium, National Council on Compensation Insurance.
8/89	Visiting Professor, Environmental Law. Vermont Law School.
11/89	Moderator, "Managed Medical Care." Workers Compensation Conference Dialogue for the Nineties. National Association of Manufacturers and Alliance of American Insurers, Baltimore, MD.
12/89	Speaker, "Validity of Various Immuno-Suppression Theories & Risk Assessment Criteria in Toxic Tort Cases." Pennsylvania Bar Institute Toxic Tort Litigation Seminar, Philadelphia, PA.
12/89	Visiting Lecturer, "State of the Art in the Use of Demonstrative Evidence in Proving Causation in a Toxic Tort Trial." Trying Mass Toxic Tort Cases: Demonstrations of Trial Techniques by Leading Practitioners and Jurists, American Bar Association National Institute, San Francisco, CA.
3/90	Speaker, "Utilization Review." Forum II Workers' Compensation Healthcare Cost Containment. International Workers' Compensation Foundation, Inc., New Orleans, LA.
4/90	Speaker and Panelist, Workers' Compensation Panel "Blueprint for Reform." 1990 Issues Symposium and Annual Meeting. National Council of Compensation Insurance, New York, NY.



-
- | | |
|-------|---|
| 5/90 | Speaker, "Assessing Chemical Hazards: Separating Scientific Toxicology from Regulatory Toxicology and Workers' Perceptions." American Industrial Hygiene Conference, Orlando, FL. |
| 10/90 | Speaker, "Chemical Hazards and Perceptions and the Role of the Industrial Hygienist." Industrial Hygiene Conference. American Industrial Hygiene Association, Ocean City, MD. |
| 11/90 | Visiting Lecturer, "Causation: Scientific Standards of Proof." 1990 Toxic Torts for Trial Judges. The National Judicial College, Reno, Nevada. |
| 1/91 | Speaker, "Perception of Chemical Assessment." PESA Health and Safety Conference. Public Employees Safety Association of Maryland, Baltimore, MD. |
| 1/91 | Visiting Lecturer, "Toxins and Health: Science Versus Perception." The Johns Hopkins University Applied Physics Laboratory Colloquium. Johns Hopkins University, Laurel, MD. |
| 7/91 | Speaker, "National Occupational Medicine Seminar." Eleventh Annual Conference. Cape Cod, Hyannis, MA. |
| 1/92 | Speaker, "Dose Makes the Poison." Northern Virginia Association of Occupational Health Nurses Inc., Vienna, VA. |
| 3/92 | Speaker, "Dioxin, Risk Assessment for Human Health." Talladega Medical Society, Talladega, AL. |
| 5/92 | Speaker, Interdisciplinary Panel Participant, "The Physician's Role in Indoor Air Complaints." National Coalition on Indoor Air Quality, Tampa, FL. |
| 5/92 | Speaker, "Determining Whether a Toxic Exposure Caused an Illness." American College of Occupational and Environmental Medicine. Post Graduate Seminar. Washington, D.C. |
| 11/92 | Conference Chair. "Multiple Chemical Sensitivities. The State of The Science." Co-sponsors - NMAS and the International Society of Regulatory Toxicology and Pharmacology. |
| 3/93 | Speaker, "Multiple Chemical Sensitivities. The State of The Science." RISE. Washington, D.C. |



4/93	Presenter, Chemical Labeling. Executive Enterprises. Washington, D.C.
4/93	Lecturer, "Disability Assessment in Occupational Disease." American College of Occupational and Environmental Medicine Annual Meeting. Atlanta, GA.
5/93	Speaker, "Multiple Chemical Sensitivities: The State of The Science." CSMA Mid-year meeting. Chicago, IL.
5/93	Speaker, "Multiple Chemical Sensitivities: The State of The Science." CTFA. Washington, D.C.
5/93	Speaker, "Risk Communication." American Industrial Hygiene Association. Annual Meeting. New Orleans, LA.
2/94	Speaker, "Relating Physical Complaints to Indoor Air Pollution." Occupational Safety & Health Regulation. Conference. Washington, D.C.
3/94	Speaker, "Health Effects and IAQ: An Overview." Indoor Environment '94. Annual Meeting. Washington, D.C.
6/94	Speaker, "The Policy of Risk Versus Individual Risk: Key to Effective Risk Communication." 19th Annual Conference & Exposition. National Association of Environmental Professionals. New Orleans, Louisiana.
9/94	Lecturer, "Risk Communication." US Army Center for Health Promotion and Preventive Medicine. Aberdeen Proving Ground, Maryland.
9/94	Speaker, "Public Versus Personal Risk: The Challenge In Environmental Risk Communication." Dixy Lee Ray Memorial Symposium on Science-Based Environmental Management. Temple University, Environmental Health and Safety. Seattle, Washington.
9/94	Moderator, "Faculty and Audience Discussion Regarding MCS," and Speaker, "Conference Summary." First Annual Aspen Environmental Medicine Conference. Aspen, Colorado.



-
- | | |
|-------|---|
| 10/94 | Speaker, "Indoor Air and Health." 4th Annual Virginia Occupational Health Conference. Virginia Occupational Medical Association. Norfolk, Virginia. |
| 10/94 | Lecturer, "Public Versus Personal Risk: The Challenge in Environmental Risk Communication." Environmental Health Program, Department of Public Health Sciences, University of Alberta, University of Calgary, Canada. |
| 10/94 | Speaker, "Low Level Chemical Exposures: What Do We Know? What Can We Say?" American Industrial Hygiene Association. Professional Development Conference. Annapolis, Maryland. |
| 3/95 | Speaker, "Toxic Tort Litigation from Environmental Exposure." 1995 Oregon Governor's Occupational Safety & Health Conference, Portland, Oregon. |
| 4/95 | Speaker, "Cost Effective Management of Medical/Scientific Aspects of Toxic Tort Claims." American Corporate Counsel Association Greater New York Chapter. New York, New York. |
| 5/95 | Speaker, "Perceptions: Addressing the Divergence Between Science and Communication." USF&G - Industrial Hygienist, Kansas City, Missouri. |
| 6/95 | Speaker, "Effects of Lead Poison - What It Shows and Doesn't Show." Chartered Property Casualty Underwriters Society, Baltimore, Maryland. |
| 7/95 | Speaker, "Multiple Chemical Sensitivities." "Sick Buildings." National Workers' Compensation and Occupational Medicine Seminar, Hyannis, Massachusetts. |
| 9/95 | Lecturer, "Legislative and Regulatory Activity, Regarding Multiple Chemical Sensitivities." 2nd Aspen Environmental Medicine Conference, Given Institute. Aspen, Colorado. |
| 9/95 | Visiting Lecturer, "Quantitative Risk Assessment and Tort Claims: What the Attorney Must Know." Indiana Continuing Legal Education Forum Seminar on Toxic Tort Litigation. Indianapolis, Indiana. |



-
- | | |
|-------|---|
| 10/95 | Speaker, "Environmentally Associated Symptoms (EAS): A New & More Appropriate Name for MCS." Chemical Specialties Manufacturers Association, Annapolis, Maryland. |
| 10/95 | Conference Chair. "Multiple Chemical Sensitivities: State-of-the-Science Symposium." Co-sponsors - The International Society of Regulatory Toxicology and Pharmacology, The Johns Hopkins University/NIOSH Educational Resource Center in Occupational Safety & Health and National Medical Advisory Service. |
| 11/95 | Speaker, MCS: Facts and Fantasy." 52nd Annual Science Conference. The Cosmetic, Toiletry, and Fragrance Association. Lake Buena Vista, Florida. |
| 12/95 | Lecturer, "Public Versus Personal Risk." Department of Design and Environmental Analysis, Cornell University, Ithaca, New York. |
| 12/95 | Speaker, "The Scientific Aspects and Application of Daubert Motions in Toxic Tort Claims." The Metropolitan Corporate Counsel. New York, New York. |
| 3/96 | Speaker, "Public vs. Private Risks." A&WMA's Waste Combustion in Boilers & Industrial Furnaces Conference, Kansas City, Missouri. |
| 6/96 | Speaker, "Application of a New Assessment Tool to Environmental Risk Communication: Merging Health Information with Psychosocial Approaches." National Association of Environmental Professionals, Houston, Texas. |
| 7/96 | Speaker, "Multiple Chemical Sensitivity: an Overview of Current Scientific and Medical Knowledge." ASTM 1996 Johnson Conference, Johnson, Vermont. |
| 8/96 | Speaker, "Chemical Sensitivities/Environmental Intolerance: Evaluation, Causation and The Impact of Causation Theories on Treatment Strategies." Allergy & Asthma Associates, Santa Fe, New Mexico. |
| 9/96 | State of the Art Speaker, "Illusions of Pseudoscience and Silicone Breast Implants." Aspen Environmental Medicine Conference, Aspen, Colorado. |



-
- | | |
|-------|--|
| 9/96 | Speaker, "Multiple Chemical Sensitivities/Idiopathic Environmental Intolerances." AIHA Chesapeake Section, Annapolis, Maryland. |
| 9/96 | Speaker, "Idiopathic Environmental Intolerances - Scientific Understanding and Regulatory/Legislative Status." The Soap and Detergent Association, Chicago, IL. |
| 10/96 | Speaker, "History, Medical Diagnoses and Causation Determination." Environmental Sensitivities Research Institute Educational Seminar for Attorneys and Administrators, San Francisco, California. |
| 10/96 | Speaker, "MCS-Real or Not?" Annual Association of Structural Pest Control Regulatory Officials, Santa Fe, New Mexico. |
| 11/96 | Speaker, "Proliferation of Health Allegations MCS, Allergies, Porphyria." The Carpet and Rug Institute Annual Conference, Dalton, Georgia. |
| 1/97 | Keynote Speaker, "Pesticide Health Risks: Scientific Truths & Public Perception." 1997 Delta Production Conference and AG Expo, Cleveland, Mississippi. |
| 4/97 | Speaker, "Controlled Exposure of Humans to Particulate Matter." 1997 American Petroleum Institute Meeting, Alexandria, VA. |
| 5/97 | Speaker, "Medical Issues" Lead Liability Litigation Seminar. Law Journal Seminars - Press. New York, NY. |
| 6/97 | Panelist, "Dialogue" on "Recent Advances in Control of Air Pollution." WorldNet Television, Voice of America Studio Washington, D.C. |
| 10/97 | Speaker, "Chemical Sensitivity." Leadership Texas, <i>Program of the Foundation for Women's Resources</i> . Houston, TX. |
| 11/97 | Speaker, "Multiple Chemical Sensitivity." Montgomery County Public Schools. |
| 3/98 | Speaker, "Understanding Chemical Sensitivity in the Population." NE Regional Turfgrass Conference. Providence, Rhode Island. |
| 3/98 | Panelist, "Do Mycotoxins Cause Health Effects in Indoor Environments?" AIHA Toxigenic Molds Workshop. Fairfax, VA. |



-
- | | |
|-------|--|
| 6/98 | Speaker, "How To Cross Examine a Scientist." DRI. San Antonio, TX. |
| 9/98 | Speaker, "New Injuries in the Workplace." International Association of Industrial Accident Boards and Commissions. St. Louis, MO. |
| 10/98 | Speaker, "Mold Evaluation and Remediation - How Complex Should it Be?" Association of Specialists in Cleaning and Restoration, Water Loss Institute's 3 rd Annual Conference & Exposition. Charlotte, NC. |
| 11/98 | Speaker, American College of Toxicology Annual Meeting. "Multiple Chemical Sensitivities: Distinguishing Between Psychogenic and Toxicodynamic." Orlando, FL. |
| 2/99 | Speaker, Mealey's Daubert and Expert Admissibility Conference. "The Migration of Toxins to Surrounding Areas or People." Jacksonville, FL. |
| 9/99 | Speaker, 3 rd Annual Mid-Atlantic Regional Conference on Occupational Medicine. "Multiple Chemical Sensitivity." Philadelphia, PA. |
| 9/99 | Speaker, Annual Alumni Homecoming CME Seminar, Marshall University. "Environmental Risk Communication: The Critical Role of the Clinician." Huntington, West Virginia. |
| 1/01 | Speaker, 2 nd National Sanitation Foundation (NSF) International Conference on Indoor Air Health. "Indoor Air and Health: Emphasize Health; Minimize Engineering." Miami Beach, Florida. |
| 3/01 | Speaker, 40 th Annual Meeting of the Society for Toxicology. "Odors in the Workplace: Minimizing Physical Illness; Not Satisfying Everyone." San Francisco, California. |
| 4/01 | Speaker, St. Paul Insurance Group. "Indoor Air Quality: Learning from the Unexpected." Baltimore, Maryland. |
| 4/01 | Speaker, International Society of Facilities Executives (ISFE). "Investigating Health Complaints in the Workplace." Kiawah Island, South Carolina. |
| 10/01 | Speaker, Property Loss Research Bureau (PLRB) Mold Symposium. "The Science of Mold and Health." Charlotte, NC. |



4/02	Speaker, DRI Mold Seminar. "The Science of Mold and Health." Coronado, CA.
5/02	Speaker, Property Loss Research Bureau (PLRB) Mold Symposium. "The Science of Mold and Health." San Antonio, TX.
5/02	Speaker, American Forest and Paper Association (AFPA). "The Science of Mold and Health." Washington, D.C.
5/02	Speaker, American Agricultural Insurance Company (AAIH). "Health Risks Associated with Mold Claims." Denver, CO.
6/02	Speaker, Mealey's Toxic Tort Conference. "Medical and Science of Causation Assessment." Pasadena, CA.
6/02	Speaker, Riverstone Resources. "Mold and Health/Managing the Process." Manchester, NH.
9/02	Speaker, Lorman Education Services Conference "Health-based Mold Management." Baltimore, MD.
11/02	Speaker, Armstrong World Meeting. "The Mold Crisis and Its Effects on Armstrong World Industries." Lancaster, PA.
12/02	Speaker, Mealey's Texas Mold Litigation Conference. "Mold Science Versus Mold Hype." Dallas, TX.
01/03	Speaker, Mold and Indoor Environment Conference. "Mold Medicine/Mold Hype." New Jersey Institute of Technology. Newark, NJ.
02/03	Speaker, Mealey's California Mold Litigation Conference. "Mold Science Versus Mold Hype: Confusing Diagnosis with Causation in Mold Matters." La Jolla, CA.
02/03	Speaker, Minnesota Association of Realtors. "Mold Medicine/Mold Hype and Its Impact on Realtors." St. Paul, MN.
03/03	Speaker, American Conference Institute. Fifth National Forum. "Toxic Mold Litigation." New York, NY.



05/03	Speaker, St. Louis Mold Conference. "Mold Medicine & Mold Science: Its Practical Applications for Patient Care, Remediation & Claims." St. Louis, Missouri.
05/03	Speaker, Peckar and Abranson and New York Construction News Seminar. "Mold, Fact Versus Fiction." New York, N.Y.
06/03	Speaker, "Mealey's Mold Litigation Conference." Amelia Island, Florida.
09/03	Speaker, Advances in Environmental Mold Issues in West Virginia." Charleston, West Virginia.
09/03	Speaker, Toxic Tort Litigation Conference. "Mealey's Practical Skill Series." Philadelphia, PA.
10/03	Speaker, Fall Meeting of ABA Seer. "Mold and Daubert Session." Washington, D.C.
10/03	Speaker, 2003 Annual DRI Meeting. "Debunking the Myths of Mold and the Application of Effective Defense Strategies." Washington, D.C.
10/03	Speaker, Lorman Education Services. "Advances in Environmental Mold Issues." Lexington, KY.
11/03	Speaker, AIHA/ASSE. Mold PDC. "A healthcare perspective on the effects of mold." Tyson's Corner, VA.
08/04	Speaker, ExecuSummit. Second Annual Mold and Insurance Industry ExecuSummit. "Medical Oversight in Water Incursion/Mold Management." New York, NY.
11/04	Speaker, Twenty Fifth Annual Meeting of the American College of Toxicology. "The Great Debate: Indoor Mold - Plague or Nuisance?" Palm Springs, CA.
05/05	Speaker, ACOEM's American Occupational Health Conference (AOHC). "Occupational Medicine: From Clinic to Courtroom." Washington, D.C.



-
- | | |
|----------------|---|
| 02/06 | Speaker, Lorman Education Services. "Indoor Air Quality." Telephone Conference. |
| 06/06 | Speaker, Mealey's Lead Litigation Conference. "The Medical Debate over What Levels of Lead Exposure are Safe." Boston, MA. |
| 10/06 | Speaker, Thirteenth Annual National Forum of Environmental and Toxic Tort Issues Conference. "Cost Effective, Provable Science in Causation Assessment for Mass Toxic Torts." Chicago, IL. |
| 04/07 | Speaker, AALNC National Education Conference. "Causation Assessment in Toxic Tort Litigation and the Critical Differences between Differential Diagnosis and the Assessment of Cause." Austin, TX. |
| 08/07 | Speaker, 10 th Annual Force Health Protection Conference. "Public Health Risk versus Personal Health Risk: The Key to Health Risk Communications" Louisville, KY. |
| 11/07 | Speaker, SETAC North America 28 th Annual Meeting. "Sound Provable Science; The Key to Assessing Public versus Private Health Risk and to Developing Helpful and Ethical Health Risk Communication." |
| 07/09
03/10 | Speaker, ASTM, "Scientific and Medical Causation." Burlington, VT
Keynote Speaker, Environmental Information Association, "Environmental Issues and Health Complaints." Austin, TX |
| 04/10 | Speaker, 2010 DRI Product Liability Conference. "A brief history of tort claims arising from alleged indoor exposures: how the past informs the future." Las Vegas, NV |
| 10/11 | Speaker, HB Conference. "Lead Toxicity." Amelia Island, FL |

PROFESSIONAL PUBLICATIONS:

1. Gots, R.E. "Study of a super-repressed mutant of E. coli B." *Undergraduate Medical Association Journal of the University of Pennsylvania*. (1966).
2. Dalal, F.R., Gots, R.E. and Gots, J.S. "Mechanism of adenine inhibition in adenine-sensitive mutants of salmonella typhimurium." *J Bacteriol*. (1966): 507.



3. Gots, R.E. "Total parenteral alimentation in newborn puppies." *Undergraduate Medical Association Journal of the University of Pennsylvania*. (1968).
4. Wilmore, D.W. and Gots, R.E. "The etiology of uric acid urolithiasis following ileostomy." *Arch Surg* 99. (1969): 421.
5. Gots, R.E. and Zuidema, G.D. "Dilation of the intrahepatic biliary ducts in a patient with choledochal cysts." *Am J Surg* 119. (1970): 726.
6. Benfield, J.R., Gots, R.E. and Mills, D. "Anomalous single left pulmonary vein mimicking a parenchymal nodule." *Chest* 59. (1971): 101.
7. Gots, R.E., Gorin, F.A. and Bessman, S.P. "Kinetic enhancement of bound hexokinase activity of mitochondrial respiration." *Biochem Biophys Res Commun* 49. (1972): 1249.
8. Gots, R.E. and Bessman, S.P. "An ultrasensitive radioassay for hexokinase." *Analyt Biochem*. (1973): 272.
9. Bessman, S.P. and Gots, R.E. "The mechanism of insulin action: mitochondrial acceptor theory." *Intrasci Chem Rep* 8. (1971): 7.
10. Gots, R.E. *The Functional Interaction of Mitochondrial Hexokinase with Sites of Oxidative Phosphorylation*. Diss. University of Southern California, 1973.
11. Gots, R.E. and Bessman, S.P. "Mitochondrial hexokinase: inner membrane location and acceptor function [abstract]." *Fed Proc*. (1973): 477.
12. Gots, R.E., Formal, S. and Gianella, R.A. "Indomethacin inhibition of salmonella, shigella, and cholera toxin mediated rabbit ileal fluid [abstract]." *Clin Res* 7. (1973).
13. Charney, A.N., Gots, R.E. and Gianella, R.A. "(Na⁺ -K⁺) stimulated adenosinetriphosphatase in isolated intestinal villus tip and crypt cells." *Biochem Biophys Acta* 367. (1974): 26.
14. Gots, R.E., Formal, S. and Gianella, R.A. "Indomethacin inhibition of salmonella typhimurium, shigella flexnari and cholera mediated rabbit ileal secretion." *J Infect Dis* 130. (1974): 280.
15. Charney, A.N., Gots, R.E. and Gianella, R.A. "Na-K-ATPase in isolated intestinal villus tip and crypt cells [abstract]." *Gastroenterol*. (1974).



16. Gots, R.E., Formal, S. and Gianella, R.A. "Salmonella mediated ileal secretion: stimulation of adenylyl cyclase, inhibition by indomethacin and possible participation of prostaglandins [abstract]." *Clin Res.* (1974).
17. Charney, A.N., Kinsey, M.D., Meyers, L., Gianella, R.A. and Gots, R.E. "Role of adrenal steroids on intestinal Na^+ - K^+ ATPase and sodium transport [abstract]." *Clin Res.* (1975).
18. Gots, R.E. "Lawyer's guide to screening medical malpractice cases." *The Retainer.* June (1975).
19. Charney, A.N., Kinsey, M.D., Meyers, L., Gianella, R. A. and Gots, R.E. " Na^+ - K^+ activated adenosine triphosphatase and intestinal electrolyte transport: effect of adrenal steroids." *J Clin Invest* 56. (1975): 653.
20. Gianella, R.A., Gots, R.E., Charney, A.N., Greenough, W.B. and Formal, S.B. "Pathogenesis of salmonella mediated intestinal fluid secretion: activation of adenylate cyclase and inhibition by indomethacin." *Gastroenterology* 69. (1975): 1238.
21. Bessman, S.P. and Gots, R.E. "The hexokinase acceptor theory of insulin action -hormonal control of functional compartmentation." *Life Sci* 19. (1975): 1215.
22. Charney, A.N., Gots, R.E., Formal, S. and Gianella, R.A. "Activation of adenylate cyclase by shigella dysenterial enterotoxin." *Gastroenterology* 70. (1976): 1085.
23. Gots, R. E. "Which of Your Patients is Likely to Sue You?" *Medical Economics*, October 18:72, 1976.
24. Gots, R.E. "Don't Panic? It's not malpractice unless..." *Medical Economics*, January 24:77, 1977.
25. Gots, R.E. "How Not to Alienate the Medical Expert." *Trial*, No. 4, April 1977.
26. Gots, R.E. "How Not to Alienate the Medical Expert." *Civil Advocates Manual*, G.O. Kornblum and J. W. Rogers, Jr., eds., University of California, (1977): 175.
27. Gots, R.E. "Malpractice. Divide and conquer tactics you should know about." *Medical Economics* 25. (1978): 48.



28. Gots, R.E. "Medical Sleuthing a Must: Evaluating Injury Cases, a Sophisticated Procedure." *The Independent Adjuster*, Chicago, IL, Spring 1980.
29. Gots, R.E. "Strategy. The medical causation defense." *For the Defense*. January (1981).
30. Gots, R.E. "Medical/scientific decision-making in occupational disease compensation." Prepared for the Crum and Forster Insurance Group, November (1981).
31. Gots, R.E. "Hypotheticals and the Expert Medical Witness." *Legal Aspects of Medical Practice* 10. (1982).
32. Gots, R.E. and Gots, B.A. "Disarming the Treating Physician: A Cost-Saving Approach to Medical Claims Defense." *Best's Review* 83. 64, May (1982).
33. Gots, R.E. "Fad Medical Claims in Personal Injury and Workers' Compensation." *The Independent Adjuster*. (1983): 29.
34. Gots, R.E. "Science, medicine, and the compensation of toxic injury claims." *Public Policy Forum*. National Association of Manufacturers, June (1983).
35. Gots, R.E. "Environmental fears and the treating physician." *The Upstate Physician*. (1983): 13.
36. Gots, R.E. "Opinion Testimony of Treating Physicians: Combatting Advocacy; Forcing Accuracy." *For the Defense* 26. (1984): 19-24.
37. Gots, R.E. "A response to H.R. 4813." Prepared for the Crum and Forster Insurance Group, March (1984).
38. Gots, R.E. "Auditing the Physician." *Business Insurance*. September (1985): 23.
39. Gots, R.E. "Medical causation and expert testimony." *Regul Toxicol Pharmacol* 6. (1986): 95-102.
40. Gots, R.E. "The science of medical causation: its application in toxic tort litigation." *For the Defense*. (1986): 4.
41. Gots, R.E. "Medical causation and expert testimony." *Causation and Financial Compensation: Conference Proceedings*, February 20-21, 1985, Washington,



-
- D.C. Washington, DC: The Institute for Health Policy Analysis, Georgetown University Medical Center, (1986): 215-23.
42. Gots, R.E. "The science of medical causation: its application in toxic tort litigation." *DRI Toxic Tort Litigation* 4. (1986): 18-27.
 43. Gots, R.E. "Workers' Compensation: The Last Bastion of the Open Medical Checkbook." *IAIABC Journal*. (1987): 35-40.
 44. Gots, R.E. "Medical Cost Containment in Workers Compensation: Controlling Excesses in Tests and Medical Care." *National Council on Compensation Insurance*. (1987).
 45. Gots, R.E. and Gleeson, J.G. "Understanding and Defending Claims of Increased Risk of Contracting Disease." *DRI Damages and Jury Persuasion* 6. (1987):54-69.
 46. Gots, R.E. "Scientific and legal approaches to proof of causation in tort cases." *The 3rd Chemical Congress of North America: The Book of Abstracts*. Washington, D.C.: The American Chemical Society, (1988).
 47. Gots, R.E. "Medical Claims Flay Casualty Insurers." *National Underwriter*, pp. 86-89, September (1989).
 48. Gots, R.E. "Workplace Illness: What We Know Versus What is Believed." *Convention Proceedings, IAIABC*, September 17-20, 1989, Baltimore, MD. International Association of Industrial Accident Boards and Commissions. (1989): 226-240.
 49. Gots, R.E. "Immune system dysfunction and toxic tort claims." *Toxic Tort Litigation*. Harrisburg, PA: Pennsylvania Bar Institute, (1989): 88-133.
 50. Gots, R.E. "Risk assessment in tort litigation." *Toxic Tort Litigation*. Harrisburg, PA: Pennsylvania Bar Institute, (1989): 85-7.
 51. Gots, R.E. "Scientific truths versus legal truths in toxic tort litigation." *Trying Mass Toxic Tort Cases: Demonstrations of Trial Techniques by Leading Practitioners and Jurists*, ABA National Institute, Nov. 9-10, 1989, Washington, DC and Dec. 14-15, 1989, San Francisco, CA. Chicago, IL: ABA Division for Professional Education, (1989): 187-207.
 52. Gots, R.E. "Medical surveillance in hazardous waste claims." *Hazardous Waste and Toxic Torts Law and Strategy* 5. (1989): 3-5.



53. Gots, R. "Managed medical care." *Highlights of National Conference on Workers Compensation, Dialogue for the Nineties*, November 8-9, 1989, Baltimore, MD. Baltimore: National Association of Manufacturers and Alliance of American Insurers. (1989): 20-3.
54. Gots, R.E. "Applying the brakes to medical casualty costs." *Best's Review* 90:10, 1990.
55. Gots, R.E. "Assessing chemical hazards: separating scientific toxicology from regulatory toxicology and workers' perceptions." *Abstracts, American Industrial Hygiene Conference: Industrial Hygiene in the World of Tomorrow*. May 13-18, 1990, Orlando, Florida. American Industrial Hygiene Association and American Conference of Governmental Industrial Hygienists, 1990. p. 20.
56. Gots, R.E. "Medical monitoring following chemical exposures." *For The Defense*. November (1990): 22-26.
57. Gots, R.E. "Deciphering the sick building syndrome." *Builder and Contractor* 39. (1991):38-9.
58. Gots, R.E. "Proving causes of illness in environmental toxicology: 'sick buildings' as an example." *Fresenius Environ Bull* 1. (1992): 135.
59. Gots, R.E. "Address fears, suppress chaos." *Public Risk* 6. (1992): 24-7.
60. Gots, R.E. "Hypothesis and practice: autointoxication and MCS." *Regulatory Toxicology and Pharmacology* 18. (1993): 2-12.
61. Gots, R.E. and Hamosh, T.D. "MCS: A symposium on the State of the Science." *Regulatory Toxicology and Pharmacology* 18. (1993) 61-78.
62. Gots, R.E. "Risk communication: a few observations from a physician/toxicologist/ communicator." *Hazardous Substances and Public Health*. (1994): 6-7.
63. Gots, R.E. "Public versus personal risk: the challenge in environmental risk communication." *Technology: Journal of The Franklin Institute* 331A. (1994): 59-65.
64. Gots, R.E. "Emphasize health to maximize problem solving." *Skylines* 20. (1995): 14-17.



65. Gots, R.E. "Multiple chemical sensitivities - public policy." *Journal of Toxicology - Clinical Toxicology* 33 (2), 111-113 (1995).
66. Gots, R.E. "Multiple chemical sensitivities: distinguishing between psychogenic and toxicodynamic." *Regulatory Toxicology and Pharmacology* 24. (1996): S8-S15.
67. Gots, R.E. "Multiple chemical sensitivities." *Hippocrates' Lantern* 4:3 (1997).
68. Light, E. and Gots, R.E. "Mold revisited a rebuttal" *Cleaning & Restoration*. July (1998): 22-32.
69. Gots, R.E. and Lees-Haley, P. "Health complaints in office workers." *Hippocrates' Lantern* 5:4 (November 1998).
70. Gots, R.E. "Evidence-based medicine and the independent medical examiner." *Hippocrates' Lantern* 6:2 (June 1999).
71. Gots, R.E. "Forensic needs versus patient health in chemical exposure claims." [abstract] *The Forensic Examiner* 9:30(#6) (January/February 2000).
72. Gots, R.E. and Pirages, S.W. "Multiple chemical sensitivities: Psychogenic or Toxicodynamic origins." *International Journal of Toxicology* 18. (1999):393-400.
73. Lees-Haley, P., Williams, C.W., Gots, R.E., and Pirages, S.W. "The neuropsychology and toxicology of residential exposure." *Journal of Forensic Neuropsychology* 1:4 (1999): 21-43.
74. Robbins, C.A., Swenson, L.J., Nealley, M.L., Gots, R.E., and Kelman, B.J.. "The health effects of mycotoxins in indoor air: A critical review." *Applied Occupational and Environmental Hygiene* 15:10 (2000): 773-784.
75. Gots, R.E. and Hamilton, D.V. "Multiple chemical sensitivities: An iatrogenically perpetuated disorder and example of the nocebo effect." *The Scientific Review of Alternative Medicine*. 5:1 (2001): 39-44.
76. Gots, R.E. and Juberg, D.R.. "Management of environmental health concerns in institutional settings." *Journal of Healthcare Safety, Compliance & Infection Control*. 5:2 (2001): 54-56.
77. Gots, R.E. "Mold and mold toxins: the newest toxic tort." *Journal of Controversial Medical Claims*. 8:1 (2001):1-8.



78. Gots, R.E. "Indoor Air and Health: Emphasize Health: Minimize Engineering." *Proceedings of the Second NSF International Conference on Indoor Air Health*. January 29, 2001: 5-15.
79. Gots, R.E. "Odors in the Workplace: Minimizing Physical Illness: Not Satisfying Everyone." *The Toxicologist, An Official Journal of the Society of Toxicology Supplement*. 60:1 (2001): 136.
80. Gots, R.E., and S.W. Pirages. "Indoor health problems: a sound process for resolution." *Facility Management Journal, a publication of the International Facility Management Association*. July/August 2001: 45-48.
81. Gots, R.E. and S.W. Pirages. "Mold as Toxin." *COLUMNS-Mold*, March 2002.
82. Gots, R.E. and S.W. Pirages. "Toxic Mold – A Misnomer." *IAQ Applications/Spring* 2003.
83. Gots, R.E., Layton, N.J. and S.W. Pirages. "Indoor Health: Background Levels of Fungi." *Am Ind Hyg Assoc J.*, July/August 2003: 427-38.
84. Gots, R.E. "Differential Diagnosis vs. Causation Assessment: Why They Are Separate Methodologies and How They Relate to *Daubert*." *BNA, Inc., Expert Evidence Report, Analysis and Perspective*. 8 November 2004. 4(21): 580-84.
85. Gots, R.E. "From Symptoms to Liability: The Distinct Roles of Differential Diagnosis and Causation Assessment." *For the Defense*. July 2005. 24-30.
86. Weiner, H.M., Gots, R.E., Hein, R.P. "Medical Causation and Expert Testimony: Allergists at the Intersection of Medicine and Law." *Current Allergy and Asthma Reports*. December 2012.

POPULAR ARTICLES

1. Gots, R.E. "The long life diet." *Family Circle* 23. (1977): 14.
2. Gots, R.E. and Gots, B.A. "Drugs, coffee, alcohol, infections, and your unborn baby." *Good Housekeeping*. January (1978): 68.



3. Gots, B.A. and Gots, R.E. "Doctor's own consumer guide to surgeons." *Self*. September (1979): 72.
4. Gots, R.E. "Bendectine: the new thalidomide?" *Genesis*. April (1979): 1-2.

BOOKS/BOOK CHAPTERS:

1. Gots, R.E. *The Truth About Medical Malpractice: Patients' Rights, Doctors' Rights*. New York: Stein and Day, 1975.
2. Gots, R.E. and Gots, B.A. *Caring for Your Unborn Child*. New York: Stein and Day, 1977.
3. Gots, R.E. and Kaufman, A. *The Peoples' Hospital Book*. New York: Crown Publishing Company, 1978.
4. Gots, R.E. "Diseases and the workplace: a framework for scientific clarification and administrative separation." *Perspectives on Healthcare Costs in Workers' Compensation*. A. Taricco and J. Greenwood, eds. LR Publications, 1992.
5. Gots, R.E. "Environmental fears and the treating physician." *Rational Readings on Environmental Concerns*. New York: Van Nostrand Reinhold, 1992. pp. 491-3.
6. Gots, R.E. "Scientific truths versus legal truths in toxic tort litigation." *Rational Readings on Environmental Concerns*. New York: Van Nostrand Reinhold, 1992. pp.493-500.
7. Gots, R.E. *Toxic Risks: Science, Regulation, and Perception*. New York: Lewis Publishing Co., 1993.
8. Gots, R.E. "Applying principles of science to Daubert motions in toxic tort claims." *1997 Wiley Expert Witness Update New Developments In Personal Injury Litigation*. 1997 pp. 1-57.
9. Gots, R.E. "Environmental risks and the clinician: informing worried patients." *Occupational, Industrial, and Environmental Toxicology*. 1st ed. Ed. M.I. Greenberg, R.J. Hamilton, S.D. Phillips. St. Louis: Mosby-Year Book, 1997 pp. 505-15.
10. Barrett, S.J., and Gots, R.E. *Chemical Sensitivity: The Truth About Environmental Illness*. Amherst, NY: Prometheus Books, 1998.



-
11. O'Reilly, J.T., Hagan, P., Gots, R., and Hedge, A. *Keeping Buildings Healthy*. New York: John Wiley & Sons, Inc. 1998.
 12. Gots, R.E. and Pirages, S.W. "Applying principles of science to Daubert motions in toxic tort claims." *2000 Aspen Expert Witness Update: New Developments in Personal Injury Litigation*. 2000. pp.1-88.
 13. Gots, R.E. "Risk Communication." in *Occupational, Industrial, and Environmental Toxicology*. Editor, Michael I. Greenberg, MD, MPH. 2003 Philadelphia: Mosby, Inc.



FOR TOXICOLOGY AND MEDICINE

EXPERT REPORT OF RONALD E. GOTS, MD, PhD, DABT

RE: *Melanie Beckemeyer v Gelco Corporation d/b/a Element Fleet Management*

INTRODUCTION

My name is Ronald E. Gots, MD, Ph.D., DABT. I am a licensed physician and Board Certified Toxicologist. As a physician and toxicologist, I have specialized in occupational and environmental medicine and toxicology for over thirty-five years. I have specialized primarily in the determination of cause and effect relationships of injuries and illnesses allegedly arising from chemical, biological (i.e., mold, bacteria and other agents) and other exposures. I have published extensively on the subject of general and specific causation, having written a number of articles and book chapters on this topic. The generally-recognized method of causation assessment in toxicology has been the topic of lectures I have given to physicians, nurses, medical students, attorneys and judges. At Georgetown University School of Medicine, I taught medical students a course on environmental toxicology in which causation methodology was emphasized. That course included a section on mold, mold toxins and their health effects.

I have been involved in hundreds of mold contamination and wet, damp building matters since the late 1980's, including courthouses, schools, other municipal buildings, assisted living facilities, hotels, homes and commercial buildings. I have evaluated health effects, prepared remediation plans, overseen remediation and functioned to insure that workers or residents not be harmed by any potential exposures. I have reviewed hundreds of scientific papers on mold, mold toxins, bacterial endotoxins, as well as other agents associated with wet, damp buildings, and their surmised, imputed and/or proven health effects and have written numerous articles on the subject. In May 2002, my firm sponsored, along with Georgetown University Medical School, a major international



symposium devoted to the health effects of mold and mold toxins. My experience consulting on indoor air quality issues affecting schools, office buildings and residences is extensive. I have overseen mold remediation activities in these structures. I have also written about indoor health and cause/effect relationships for a diverse range of environmental pollutants.

In the past thirty years, as CEO of the International Center for Toxicology and Medicine and founder of Building Health Sciences, my associates and I have been involved extensively in indoor environmental matters. I have examined over two hundred patients, occupants of residential and municipal buildings, as well as schools (both students and teachers) who believed that mold in their facilities was making them ill. I have seen the breadth of complaints, some minority of which may have been mold-related, most of which were not, but were perceived to be so by involved individuals.

I have also visited dozens of buildings—schools, homes, apartment buildings, assisted living facilities, hotels, courthouses, and other public and commercial buildings—throughout the United States in which there was water damage, mold growth and concern about possible health effects. Some of these had exuberant mold growth covering many walls; others, small spots to no observable mold growth. Thus, I have personally seen all of the extremes of water damage and resultant growth of mold. In those investigations, it was often my responsibility to assess health risks, make decisions about removing individuals from the environment and, with my colleagues, to develop remediation plans.

Consequently, I have an extensive professional background in the science of health effects from indoor environmental contaminants, the evaluation of individuals who have been in such environments and the investigation of facilities which suffered varying degrees of water intrusions. This, plus my past and ongoing review of thousands of primary research papers, position papers and books dealing with the issues at hand inform my understanding of these matters and my ability to analyze them.

Attached is a true and correct copy of my Curriculum Vitae which details my experience, training and education with regard to toxicology, the science of causation analysis, to mold and other agents associated with wet, damp spaces, and their effect upon human health. (Appendix A)



In preparing this report, I have reviewed the following materials concerning Ms. Beckemeyer:

MATERIALS REVIEWED

Medical Records

Masood Ahmad, MD (3/1/2001-12/27/2016)

H.S. Blatman, MD (5/22/2017)

Craig P. Cleveland, MD (9/22/2016-5/16/2017)

Paul S. Hoga, MD (5/8/2017)

Gary Huber, DO (5/22/2017-5/28/2018)

Kroger Little Clinic (10/21/2013-10/28/2017)

Suzanne Matunis, MD, Blue Ash Family Medicine (6/1/2011-9/25/2018)

Medical University of Cincinnati (1/1/2016-8/1/2017)

Harold Pretorius, MD (3/11-8/29/2017)

Liberty Urgent Care (12/17/2012)

Richard E. Sexton, PhD (5/5-5/18/2017)

Quest Diagnostics (3/1/2017)

Workers' Compensation First Report of Injury (10/21/2016)

Legal Documents

Complaint (10/18/2017)

Technical Data



Ecostratum Report (1/9/2018)

Gelco Record Production

Depositions

Melanie Beckemeyer (9/27/2018)

BACKGROUND OF THE CLAIM

Melanie Beckemeyer believes that mold in a company car injured her. Ms. Beckemeyer, a pharmacist, was working for Avantir Pharmaceutical when, on June 20, 2016, she first drove her company-provided vehicle. She later claimed that she noted a bad smell and, within thirty minutes of driving the vehicle, became dizzy and lightheaded. Subsequently, she also complained of sinus and ear congestion, a non-productive cough, nasal discharge, a sore throat and intermittent shortness of breath. Nearly one month after exposure to the car ended, she reported developing “brain fog.” She ascribed these symptoms to mold in the car. However, the medical records show that the symptoms are temporally disconnected from the car: she had them before, less during, and more after giving up the car. They also have clear alternate causal explanations: infections and allergies primarily. Moreover, there was no mold growth identified in the car.

The last day she drove the car was September 23, 2016. The last day she worked was October 20, 2016.

Ms. Beckemeyer states in her Complaint that she is now “disabled from employment and suffers from severe cognitive impairment and other maladies.” She reports constant symptoms of dizziness, nausea, lightheadedness, confusion and brain fog.

MEDICAL HISTORY

Between June 15, 2011 and September 18, 2015, Ms. Beckemeyer was followed and treated at Blue Ash Family Medicine by her PCP, Dr. Suzanne Matunis, for panic disorder, major depression, single episode and stress-related hypertension. She reported being tearful all the time, unable to concentrate, trouble falling/staying asleep, unwilling to get up in the morning, anhedonia, racing heart, panic attacks with chest tightness and shortness of breath. In the past, she had had situational anxiety, but denied depression as severe as she was having at this time. Tearful and distraught, she was jumping from thought-to-thought, but denied being suicidal. Her blood pressure was

Page 4



elevated, something she had experienced in the past with stress. She attributed her symptoms to work stress. She preferred taking leave and working with a therapist to taking medication. She later admitted that, while work stress was contributory to her symptoms, she had significant anxiety related to her mother's and her dog's illnesses.

Medication successfully controlled her blood pressure, but she developed palpitations when she had to deal with work. By June 27, 2011, she felt anxious frequently, had decreased energy and problems with future planning, functioning and concentrating due to stress. She remained off work because of continued anxiety, poor focus and stress from contact with HR. Therapy was begun in mid-July 2011, but difficulty focusing and concentrating continued. She felt she could not return to work and be productive. By early August, she was still experiencing occasional panic attacks, had low energy, very poor concentration and focus. Minimal stress caused her to cry and feel anxious. She remained off work. She was self-administering health food supplements which she thought were helping.

By September 8, 2011 Ms. Beckemeyer's leave of absence was up: her employer indicated she had to return to work or she could be terminated. She claimed she was having panic attacks nearly daily and was unable to return to work. Her panic attacks worsened when she thought about returning to her previous work environment. Celexa, an anti-depressant also used to treat anxiety, was begun September 8, 2011.

In mid-September, she began another form of therapy: EMDR [eye movement desensitization and reprocessing]. As of September 26, 2011, she had a new job offer for the end of October. Despite feeling anxious with occasional heart racing, she was having no full-blown panic attacks. Celexa was continued through September 2015. Ms. Beckemeyer did not return for anxiety/depression or any other complaints after this visit for fifteen months.

Ms. Beckemeyer's medical records available to me begin with an allergist's appointment in 2001. She was 43 years old and reported she had had allergies for at least ten years. Her symptoms, most prominent in summer and fall, included sneezing, nasal congestion and discharge, itchy/teary eyes, mouth-breathing/snoring, sinus infections, irritability, hoarseness and headache. She had a past history of surgery on her turbinates, a septoplasty [nasal surgery], as well as surgery on the balance mechanism of her inner left ear. Examination revealed boggy, swollen nasal mucosa and enlarged (3+-4+) turbinates:all evidence of allergies. She was treated with allergy medication: pills, nasal



spray and allergy shots (immunotherapy). She continued throughout the available records to use nasal allergy spray.

Allergy testing in 2004, by her allergist Dr. Ahmad, included only intradermal testing, not prick/puncture testing. She had 2+ [intradermal] reactions to cat, dog and mold mix 2 (*Phoma*, *Fusarium*, *Mucor*, *Pullularia*, *Rhizopus*). On both visits, Ms. Beckemeyer reported having a pet dog. She had a slight reaction to mold mix 1 (*Alternaria*, *Asp. niger*, *Helminthosporium*, *Pen. Notatum*). Subsequently, she had allergy shots, after which her allergy symptoms improved.

Sore throat, nasal congestion, dry cough, chest tightness and mild laryngitis were diagnosed in August 2011, as bronchitis, by Dr. Matunis. An antibiotic was prescribed.

In April 2012, Ms. Beckemeyer, 53 years old, complained to Dr. Ahmad of sinus pressure and pain, as well as fatigue. Her eyes were red, the nasal mucosa was boggy, her turbinates were 4+ enlarged and there was nasal discharge evident. She had sinus tenderness and a red throat. Acute sinusitis was diagnosed and treated with antibiotics and steroids.

Dr. Ahmad saw her again in December 2012. She complained of chest congestion, post nasal discharge and a stuffy [congested] nose. He noted her past history of allergic rhinitis, allergy shots, recurrent sinus infections, runny nose, nasal congestion and sinus pressure. She was taking Cromolyn and Nasonex for her upper respiratory allergies, as well as Metformin for diabetes. Her nose had yellow discharge and there was drainage in the back of her throat (PND). She gave a history of asthma. Dr. Ahmad prescribed an antibiotic and Proventil inhaler. Allergic conjunctivitis and acute pharyngitis [sore throat] were diagnosed.

Five days later she was seen in Urgent Care complaining of constant, moderately-severe central chest congestion, with a feeling of pressure and a mildly-productive cough. Chest X-ray was normal. Acute bronchitis was diagnosed and treated with antibiotics, Prednisone and a cough suppressant.

After an absence of fifteen months, Ms. Beckemeyer, on December 18, 2012, returned to see Dr. Matunis, complaining that she had been returning from a trip when someone on the plane was coughing. Four days later, she had developed a cough, chest and sinus



congestion, sore throat and malaise. She had seen her allergist, had taken two courses of antibiotic, but was still ill. Bronchitis was diagnosed and a new antibiotic prescribed.

One year later, in December 2013, Dr. Matunis performed a yearly physical on Ms. Beckemeyer. The latter reported that she had had elevated blood pressure a few weeks earlier and started taking an old prescription given her for hypertension related to stress. She noted her pulse was in the “upper 80s,” when stressed. The examination was normal. Dr. Matunis wrote a new prescription for an anti-hypertensive, as well as for Celexa, an anti-depressant/anxiolytic.

At the University of Cincinnati Medical Center, Ms. Beckemeyer, now 56 years old, was seen for follow up of left ear reconstructive surgery in June 2014. She had age-related, bilateral sensory-neural hearing loss. Gait, posture and positional function were normal. However, in September 2015, she complained that she was “woozy” and had vertigo following neck manipulation. She was treated with Valium and a steroid dose pack.

On December 15, 2014, Dr. Matunis again saw the claimant for a yearly physical. She was stressed by her mother’s illness [myelodysplasia] and was looking for a new job. She had stopped taking blood pressure medication because her elevated pressure had “resolved.” Her past medical history was noted to include polycystic ovaries and

Meniere’s disease, a condition associated with vertigo and imbalance. Examination was normal; Dr. Matunis again prescribed Celexa and Nasonex.

Two weeks of ear pressure, left more than right, nausea, ear-fullness and popping, feeling off-balance and “swimmy,” along with nasal congestion caused Ms. Beckemeyer to see Dr. Matunis on September 18, 2015. She had a past history of left eustachian tube reconstructive surgery. Examination revealed no ear findings, but there was boggy mucosa in the nose with mucoid discharge. A Medrol [steroid] dose pack, Nasacort and Celexa were prescribed. Ms. Beckemeyer requested a letter since her employer required her to take a pre-arranged shuttle from the airport which made her nauseated.

After undergoing neck manipulation for TMJ symptoms, Ms. Beckemeyer developed nausea and vertigo, a severe type of dizziness. She consulted her physician at the University of Cincinnati. She was advised to discontinue using Flonase and begin Nasonex. Celexa was continued.



Ms. Beckemeyer returned to Dr. Matunis in January 2016, for left hip pain after slipping on ice. Ibuprofen and a Medrol Dosepak were prescribed for lumbar tenderness and spasm. She continued seeing her physician for this problem through mid-September 2016.

Avanits Pharmaceuticals hired the claimant on April 25, 2016. She first drove a company-provided car on June 20, 2016. On June 28, 2016, Ms. Beckemeyer was seen at Kroger Little Clinic for sinus and ear congestion, headache, non-productive cough, chest tightness, nasal discharge and a sore throat for three days. These symptoms were the same ones she repeatedly reported to Dr. Ahmad as typical of her past history in 2001 and 2012. Later, in the context of this claim, she claimed that she had noted a bad smell and, within thirty minutes of driving the company vehicle (June 20), she became dizzy and lightheaded. That is not the history she related to the medical provider on June 28. In this contemporaneous record, there was no mention of becoming symptomatic when she first got the car or of noticing any odor in the car.

She acknowledged, on this June 28 visit, that she had sick contacts at home (in other words, a source of infection). She gave a history of seasonal allergies, multiple ear surgeries and post nasal drip (PND). Examination revealed clear fluid behind her eardrums, mucoid nasal discharge, inflamed mucosa, swollen nasal turbinates and maxillary [sinus] tenderness. Her throat was normal; her lungs were clear. Viral sinusitis

was diagnosed. Sudafed and Prednisone were prescribed. Thus, symptoms she later ascribed to the car were, eight days after she got it, diagnosed as a viral sinus infection which was clearly the correct diagnosis. There is not a hint of evidence or reason to believe from this record that exposure to mold in the car (even if there were mold in the car) played any contributory role.

Dr. Matunis saw Ms. Beckemeyer September 13, 2016, for an upper respiratory infection that had been present for three-to-four days. Once again, this was a new-onset event, three months into her use of the car. There was no mention of the car or of ongoing symptoms. Her nasal mucosa was boggy, with a mucoid discharge. The physician diagnosed acute nasopharyngitis and prescribed an antibiotic to be taken if, after ten days, the symptoms had not improved or had worsened. Once again, Ms. Beckemeyer had another infection, having nothing whatsoever to do with the car. She later ascribed the symptoms of that infection to her perception that contamination in her car was responsible.



Nine days later, September 22, 2016, Ms. Beckemeyer appeared at the office of Dr. Ahmad, her allergist. She complained of symptoms similar to those she had described in June, at the Kroger Little Clinic and in mid-September to Dr. Matunis: nasal congestion, intermittent headache, some throat clearing with excessive post nasal drip (PND), severe sore throat. He diagnosed perennial allergic conjunctivitis and allergic rhinitis due to pollen. She did not tell the allergist that she was seen nine days earlier and diagnosed with an upper respiratory infection. Nor did she report that she was given an antibiotic which she did not take. She told him that all of these symptoms began after exposure to mold, not that she had several infections which had nothing to do with mold; or, that neither mold related or water damage, nor the putative leak causing it was found when her car was evaluated by Performance Toyota.

All of these symptoms, she now said, had begun after being exposed to a car with mold. However, she acknowledged a long history of perennial allergy symptoms, of variable frequency during each month. She also said that she had symptoms at home, at work, at other people's homes and outdoors, as well as in her car. Other symptom triggers included upper respiratory infection, weather changes and dusting. She denied having had allergy testing (she actually did in 2004) and noted she had pet dog(s). She later tested positive to dog on allergy testing. Her few physical findings were similar to her previous visits in June and September: puffy eyes, boggy, swollen, irritated nasal mucosa; her throat was normal. Qnasl, a steroid spray for allergies was prescribed, as was a saline nasal spray. Dr. Ahmad diagnosed allergic rhinitis due to pollen, perennial

allergic conjunctivitis; he noted symptoms of nasal congestion, post nasal drip, dizziness and effects of change in ambient water pressure on ears.

Ms. Beckemeyer later told Dr. Matunis that the last day she drove the car was September 23, 2016. She said that her allergist, Dr. Ahmad, had told her that it "was very difficult to get rid of [mold] and no chemical way to kill it." He allegedly told her not to drive the car.

On October 13, 2016, Ms. Beckemeyer was seen at the University of Cincinnati. She reported having had "excessive mold exposure" over the summer from a car leak which had caused her to develop respiratory issues. She had gotten rid of the car two weeks earlier; she was feeling better, but her symptoms were not gone. She had a history of Meniere's disease, for which she had had a left endolymph shunt and a tube that had been in her ear for more than twenty years. That disorder produces, among other things,

Page 9



dizziness. Since 2004, she admitted having some motion sensitivity which, she claimed, had been “exacerbated by this mold exposure.” She denied ear pressure, despite having reported ear congestion and ear pressure in June and September 2016, respectively. She was taking Qnasl for some PND and “persistent facial pressure,” a brand new complaint. Her past history included immunotherapy, after which her allergy symptoms had abated. Due to fly in a few days for her job, Ms. Beckemeyer was concerned about doing so until her symptoms improved; the physician concurred.

Dr. Ahmad saw her the following day, October 14. She was complaining of nasal congestion, sneezing, some dizziness/lightheadedness, some ear pressure (denied, just the day before) and a frontal, mid-facial headache. She denied confusion, paresthesias, dysarthria or gait instability, cough, shortness of breath, wheezing, anxiety or depression. Physical findings on examination included red, puffy eyes, circles, as well as boggy, swollen, irritated nasal mucosa. Sinuses, throat, ear, voice, tonsils and chest were all normal. Zyrtec, an allergy pill and Singulair were prescribed. Dr. Ahmad noted nasal congestion, PND, moderate persistent asthma, food allergy and dizziness. This was now several weeks after she had stopped driving the car. This makes it quite clear that ongoing allergic symptomatology was related to factors having nothing to do with the car. If one is exposed to an allergen source, then eliminating that exposure ends the allergic response. The fact that the allergic symptomatology did not abate after the car was gone means the car was not responsible.

When Dr. Matunis saw Ms. Beckemeyer on October 18, 2016, the history provided was that the car she had been provided for work had had mold which caused nasal congestion, ear pressure, balance problems, brain fog (a new complaint), mild headache and facial congestion. She believed she wasn’t getting better. The only physical findings were boggy nasal mucosa with a mucoid discharge. Dr. Matunis, based on the information given her, diagnosed allergic rhinitis due to mold. A steroid dose pack was prescribed. These symptoms, it is worth noting, were similar to ones reported throughout her medical records, beginning before she ever drove the company car in 2016, and associated with her general allergy symptoms, sinusitis and upper respiratory infections.

Ms. Beckemeyer stopped working on October 20, 2016. A First Report of Injury to Ohio’s Bureau of Workers’ Compensation listed the diagnosis of Allergic Vestibulitis due to repeated mold exposure in company vehicle. The basis for this claimed diagnosis is not



clear. It was not listed as a diagnosis by any physician in any of the medical records available for this review.

When she returned to Dr. Ahmad on October 24, Ms. Beckemeyer now complained of, “lots of dizziness symptoms, some sinus pressure, nasal congestion, left ear full, lots of post nasal drainage.” Her physical findings were similar to previous examinations: puffy eyes, boggy, swollen, irritated nasal mucosa. She had new symptoms of tenderness to palpitation of moderate degree over the right frontal and of significant degree over the left frontal sinuses. Singulair tablet and saline nasal spray were prescribed. Xyzol was discontinued. Once again, this was a month after the car was gone. Clearly, these allergic symptoms and manifestations were related to other sources of allergens, not to the car.

The same day, she was seen by Dr. Matunis, to whom she reported persistent dizziness and malaise. She had not been in the car since September 23, but her symptoms had “continued to get worse.” The Prednisone had been of “no help,” in fact, Ms. Beckemeyer thought it “may have made [her] worse.” She was “unable to stay on task,” because the dizziness affected her ability to focus. Unable to drive or to fly, she had nasal congestion, no discharge, mild PND, pressure in her left ear, but no hearing loss or tinnitus, fever, chest congestion, cough or wheeze. Her “balance was off, but she had no full-blown spinning. She was taking Sudafed and Bonine, with minimal-to-no improvement. Dr. Matunis noted boggy nasal mucosa with mucoid discharge. She prescribed Anti-Vert and advised Ms. Beckemeyer to stay off work for two-to-four weeks.

On October 27 and 28, 2016, Ms. Beckemeyer returned to the University of Cincinnati Medical Center for worsening of her motion sensitivity which had been present since her

2004 ear surgery. She attributed the worsening to mold exposure. Despite changing vehicles, her symptoms of lightheadedness, imbalance, nausea, constant foggy thinking and some sensitivity to loud sounds. Her recent sinus congestion, intermittent left ear pain and popping had always been successfully managed with Dramamine, pseudoephedrine, but these were no longer working. Vertigo, headache and blurred vision were denied. Examination of the ears revealed poor movement of the left eardrum and normal central nervous system examination. VNG and VEMP testing were ordered. Again, she made the attribution to mold in the car, but that was clearly incorrect. She had not been in the car for over a month. Moreover, the car had never had a mold issue.



Dr. Ahmad saw Ms. Beckemeyer on October 31, 2016. She reported increased symptoms of dizziness, lightheadedness, a lot of throat clearing and some headaches. Nasal congestion, a runny, stuffy nose and sneezing were also described. She denied confusion, but said brain fog was still present. The only findings were puffy eyes with circles and boggy, swollen, irritated nasal mucosa. Qnasl was continued and hydroxyzine, an antihistamine, was prescribed. He again diagnosed allergic rhinitis due to pollen and perennial allergic conjunctivitis. The same day, Dr. Ahmad wrote a letter stating, "Ms. Beckemeyer is under my care and is experiencing constant dizziness, nausea and brain fog. Motion exacerbated these symptoms. I advise she not return to work until the situation is resolved."

Dr. Gamm, the Medical Review Officer for Ohio's Bureau of Workers' Compensation, on November 3 2016, denied Ms. Beckemeyer's Workers' Compensation Claim. He noted that "the medical documentation supported her claim of allergic rhinitis and allergic conjunctivitis due to mold as being directly related to the 9/22/2016 injury. However, nasal congestion, post nasal drip and dizziness are symptoms, not diagnoses and should not be considered. Finally, medical documentation did not support any additional conditions related to this claim nor any relevant pre-existing conditions."

The Workers' Compensation form completed by Ms. Beckemeyer on November 15, 2016, noted that she had last worked on October 20, 2016. She had left work because of "repeated exposure to mold in company-provided vehicle." She listed the following symptoms as being related to that exposure: "dizziness, vertigo [new], nausea, motion sickness, brain fog, lightheadedness, confusion, spacey [new], impaired memory [new] and cognition." Thus, seven weeks after use of the care ceased, she reported several brand new complaints. Brain fog had first been reported more than three weeks after she stopped using the car in which she claimed mold exposure occurred.

For the last time, on December 13, 2016, Ms. Beckemeyer saw Dr. Ahmad. She told him she was "doing well, in general." She had occasional itchy eyes and occasional dizziness, but no sinus symptoms, no ear pressure, headaches, chest congestion or coughing. At times, she had mild-to-moderately severe nasal congestion and somewhat increased PND. Her eyes still appeared puffy; her nasal mucosa was enlarged and bluish in appearance. He again diagnosed Allergic rhinitis due to pollen, perennial allergic conjunctivitis, nasal congestion, PND, dizziness and added food allergy. Allergy testing was performed on this visit. Skin prick testing was 3+ for two molds: *Epicoccum*



nigrans and *Fusarium*. 1+ for soy and 1-2+ for peanut. Intradermal testing, a form of allergy testing less likely to be associated with clinical symptoms, was 1+ for cat, dog and ragweed; she had no reaction to Mold mix 1. She had a 2+ reaction to Mold mix 2 which included the two molds she was sensitive to on prick testing. It should be noted that neither of these molds to which she tested positive are commonly associated with interior wet spaces. Others are to which she did not test positive.

On December 13, 2016, Ms. Beckemeyer saw Craig Cleveland, MD. She told Dr. Cleveland that her dizziness and motion sickness were improved. She had no sinus, vision or hearing problems, no headaches or motor difficulties. For the first time, in the available records she described cognitive problems: word finding difficulties, slow thought, being forgetful, all of which, she complained, had not significantly changed or improved. She had not reported these previously to any physician whose records I have. She claimed that she had increased her Nystatin (anti-fungal medication) to twice per day. I have no record of who gave her this. Dr. Cleveland noted that this appointment was a Workers' Comp follow up visit, but there is no prior record from his office provided. He diagnosed allergic conjunctivitis, allergic rhinitis, labyrinthitis, motion sickness, confusion, cognitive impairment.

From this point on, Ms. Beckemeyer saw standard medical practitioners only rarely. She visited her Primary Care physician, Dr. Maturnis (2/1/2017, 4/12/2017, 6/19/2017, 12/28/2017, 3/26/2018 and 9/25/2018) for shoulder pain and urinary tract complaints. The available medical records end September 25, 2018, more than two years after Ms. Beckemeyer last drove the company car she believed contained mold. She complained about sinus and ear pressure, mild vertigo and feeling her balance was "off all the time." She claimed that these had been problematic since her "mold allergy [exposure]." The only findings were in her nose: boggy tissue and mucous discharge. Although Ms. Beckemeyer erroneously believed she was still experiencing symptoms from her long past car exposure, Dr. Matunis recognized allergy-related findings and diagnosed "seasonal allergic rhinitis due to pollen." An exposure, even to something to which she was actually clinically allergic, could not, two years after that exposure ended, cause continuous, protracted symptoms.

Ms. Beckemeyer also consulted her ear surgeon at University of Cincinnati and was evaluated there by a neurologist, Dr. Shatz, who also tested her for her multiple cognitive complaints. She had become convinced, by her internet research and non-standard



practitioners that she had an “inflammatory process” in her brain, as well as her body, related to mold.

Detailed, standard neurological evaluation at the University of Cincinnati by Dr. Shatz in August 2017 revealed a completely normal neurological physical examination, although she did comment that Ms. Beckemeyer was “very distressed about the symptoms” she believed were caused by mold exposure and she was “concerned about future employment.” Despite repeated complaints of word-finding difficulties, examination found that she had normal rate, rhythm and clarity of speech, as well as verbal agility. She had both intact grammatical expression and comprehension. Language was fluent, without any word-finding difficulty, blocks or circumlocutions. Testing was conducted for the complaints of “cognitive impairment with memory loss.” Dr. Shatz found only “mild cognitive impairment with memory” and concluded that the overall pattern of memory impairment was consistent with impaired attention and concentration, to which her admitted sleep disturbance contributed.

As noted, the majority of Ms. Beckemeyer’s medical visits and care after November 2016 was provided by non-standard medical providers who subjected her to a vast array of unnecessary diagnostic studies and treatments. The diagnoses and treatment regimens they provided were all directed at indicting mold as the cause of her complaints. Their practice patterns which are not generally-accepted or scientifically-supportable will be discussed below.

MEDICAL ANALYSIS

Nasal and other upper respiratory tract symptoms: allergy or infection

Although Ms. Beckemeyer has associated the onset of her symptoms to driving a company vehicle which she decided had mold, the contemporaneous medical records reveal that during this period she was diagnosed with upper respiratory infections, the actual cause of her symptoms. On June 28, 2016, she had sinus tenderness, inflamed nasal mucosa and discharge. Viral sinusitis was diagnosed and treated with steroid medication.

It appears that these symptoms resolved with the prescribed treatment because when she saw her primary care physician, Dr. Matunis, one week later on September 7, for



back pain, stiffness and foot numbness, she reported no complaints like those described in June. Moreover, she was not taking any medications for those kinds of complaints.

On September 19, 2016, Dr. Matunis diagnosed her with acute, that is, recent onset, naso-pharyngitis. Her complaints were new: she reported a three-to-four day history of an upper respiratory infection, nasal congestion, ear and sinus pressure, with nasal discharge and a morning cough. An antibiotic was given only to be taken if her symptoms did not improve or got worse within ten days. Thus, once again, while she had the car she had a non-car-related cause of symptoms. Moreover, those had not been continuous with the car usage, as she claimed; rather, they were intermittent and acute.

Three days later, on September 22, she saw her allergist, Dr. Ahmad and reported a brand new history and set of symptoms. She claimed that “nasal congestion, some lightheadedness [new complaint], intermittent headaches and some throat clearing, severe sore throat and excess post nasal drip” had “all begun after being exposed to a car with mold.” A steroid spray was prescribed. She later said that Dr. Ahmad told her to get rid of the car. The last day she drove it was September 23, 2016. Her last day of work was October 20, 2016.

Allergic symptoms arise when the allergic individual is exposed to the allergen to which she is sensitive. The presence of the allergen causes the immune system of an allergic person to react. Once the allergen is removed, stimulation of the immune system and the related symptoms cease. She admitted that she had a long history allergy symptoms throughout the year, with symptoms during each month. Ms. Beckemeyer tested positive to two molds in December 2016: *Epicoccum nigrum* and *Fusarium*, neither of which is identified as one of the water-loving molds commonly found in damp situations, as is alleged here. Both of these mold/fungi are found in air and are plant pathogens: the former grows on the surface of plant leaves, the latter is found in soil. Both can be transported on pet fur, shoes and clothing into the indoor environment and are commonly found in homes and buildings. Allergic reactions to these molds are just as, or more likely, to occur at home and out-of-doors than in a car. If, as alleged, the car was the source of her allergic symptoms, they should have improved after her exposure to the source ended. If Ms. Beckemeyer’s symptoms were allergic in nature, as Dr. Ahmad’s findings, diagnosis and treatment confirm, then the continued persistence of her eye, nose, throat and sinus complaints would be typical and confirm her continued exposure to allergens in her home, possibly her workplace and the outdoor environment. The fact



that the car was long gone yet symptoms continued mitigates against anything causal associated with the car.

Neurological complaints: lightheaded, dizzy, balance off, motion sickness, vertigo

Ms. Beckemeyer had longstanding problems with her middle ear and her balance beginning at least in 2004. She related that motion sensitivity, present since 2004 ear surgery, had been “exacerbated by excess mold exposure.” Lightheadedness was reported on October 14 and 18. On October 14, dizziness was first mentioned.

By October 24, “lots of dizziness,” was described to Dr. Ahmad; Ms. Beckemeyer had not been in the car for over one month. The same day, she told Dr. Matunis that she had “persistent dizziness and malaise [new complaint]. Symptoms continued to worsen.” On October 27, she claimed that her “dizziness was getting worse.” When her subjective complaints of dizziness and imbalance were evaluated on October 28, at the University of Cincinnati, “no specific peripheral vestibular problem was found,” but abnormal tympanic membrane findings were present. Vestibular testing was ordered, but MS. Beckemeyer did not complete it. Increased dizziness was again related on October 31.

Although Ms. Beckemeyer later claimed dizziness had begun thirty minutes after she first drove the car in June, the medical records confirm lightheadedness was first reported on September 22, months after she got the car and dizziness on October 14, 2016, after she gave it up. Thereafter, increasing and persistent dizziness continued to be described, as well as imbalance and vertigo, a unique type of dizziness. Even if dizziness were related to some allergic reaction to the car, it would not begin, then worsen and persist weeks, months and over a year after that exposure had ended, as is claimed here. The most likely explanation for ongoing dizziness/vertigo was related to her longstanding motion problems which followed her endolymphatic surgery in 2004.

Neuropsychiatric symptoms: brain fog, confusion, disorientation, memory loss, trouble following conversation, trouble initiating tasks, word-finding difficulty, difficulty focusing, anxiety

Another of Ms. Beckemeyer’s persisting complaints is “brain fog.” Although she attributes this to mold in the car, it wasn’t reported until October 18, 2016, three weeks after she had ceased driving that car. “Brain fog” suggests unclear or confused thinking, yet she



specifically denied any symptoms of confusion on October 14, to Dr. Ahmad. The term “brain fog” is a symptom commonly associated with anxiety.

The first time “impaired memory” appears as a claimed symptom is in the Workers’ Compensation form Ms. Beckemeyer completed November 15, 2016. The first time “word-finding” as a problem is reported is December 13, 2016, in Dr. Cleveland’s medical record. There is no conceivable pathophysiological mechanism that could explain the development of any of these new symptoms, long after the car exposure ended or their persistence more than a year later, as is claimed. There is also no conceivable way that mold could ever cause such disorders. There are no recognized, accepted brain effects of indoor environmental mold exposure of the type experienced by Ms. Beckemeyer.

The absence of any organic brain injury affecting cognition (memory, speech, language, thought) is confirmed by the neurological evaluation and testing performed at the University of Cincinnati in August 2017, by Dr. Shatz. Ms. Beckemeyer admitted to mildly reduced overall sleep time; Dr. Shatz felt that this sleep disturbance could contribute to her distractibility, affecting attention/concentration and, therefore, memory. She specifically commented that the test result findings were non-neurodegenerative and attributable to the reported sleep disturbance. Ms. Beckemeyer admitted to Dr. Shatz that she was anxious and distressed, both of which are well-recognized as affecting one’s ability to attend and concentrate, thus impacting memory. The family history of dementia in Ms. Beckemeyer’s mother and two maternal uncles was recorded by Dr. Shatz, but she did not diagnose any neurodegenerative disorder or Alzheimer’s. Brain MRI revealed mild-to-moderate white matter changes consistent with aging and chronic small vessel ischemia, most often seen as a manifestation of hypertensive arteriosclerosis. The medical records chronicle frequent poor control of the claimant’s high blood pressure. There are no generally-accepted, known brain effects of mold, either through allergy or so-called “toxicity.”

It has been alleged by some of Ms. Beckemeyer’s medical providers that she is experiencing some neurological effect induced by the claimed car mold. Short of a fungal mold infection of the nervous system which she clearly does not have, such an effect does not occur.



As noted earlier in this report, Ms. Beckemeyer sought the majority of her care after November 2016, with a series of fringe practitioners, Drs. Cleveland, Blatman, Pretorius and Huber, who encouraged her mistaken notions regarding mold and its health effects. As will become clear later in this report, their unnecessary and misleading testing, treatments and erroneous causal attributions only served to support her mistaken beliefs, hindering her ability to receive proper treatment or to recover, ultimately causing her to believe she is totally disabled and unemployable. Their diagnoses include Chronic Inflammatory Response Syndrome (CIRS), a made-up non-illness from mold and other biotoxins requiring detoxification; brain vasculitis; abnormal neuropsychological findings from “biotoxin” exposure; or, an immunological illness caused by a variety of ill-defined toxins. These “diagnoses” and causal attributions are not recognized by the standard medical community; they are neither generally-accepted nor scientific knowledge. Rather, they are the province of a small group of fringe practitioners who support vulnerable patients’ false belief systems.

ENVIRONMENTAL DATA ANALYSIS

Ms. Beckemeyer took her company vehicle to Performance Toyota on June 23, 2016. She requested the leak from the passenger side visor be fixed. According to the information in the Performance Toyota records, no specific leak is described; a damaged washer line, possibly chewed by mice, was replaced. There is no description of any mold. At her request, the HVAC evaporator was flushed and all of the water was removed from the evaporator box.

Testing of the vehicle was conducted by Ecostratum in July 2018. All levels of molds sampled in the car, in air and dust, were either low-to-non-existent and/or no different from common background levels found in non-water damaged homes. For example, the highest mold level found was 10,000 cfu/gm of carpet dust (*Penicillium*). Hicks et al studied never water-damaged homes in California. The average mold levels in carpets with low traffic were >55,000 cfu/gm of dust. Of that, approximately 12,000 was *Penicillium*. Even bedspreads had > 24,000 cfu/gm of dust. Since molds are carried into interior spaces like cars and homes on clothing and shoes, it is hardly surprising that car carpets will have mold when tested. So does all of our home carpeting. The air levels in the car, too, were well below those commonly found in indoor air (Gots et al 2003, Baxter et al 2005).



MEDICAL CAUSATION ANALYSIS

Recognized effects of mold and mold toxins (general causation)

The primary scientifically-known and generally-accepted mold effects and those of other factors associated with wet-damp spaces, relevant to this matter are discussed by the National Academies, Institute of Medicine (2004), Bush et al (2006), World Health Organization (2009), The German Society of Hygiene, Environmental Medicine and Preventive Medicine (2017) and Borchers et al (2017). They include:

- Upper respiratory allergies (like hay fever) in some allergic people which produce:
 - Red, watery eyes
 - Sneezing and runny nose
 - Stuffiness and congestion
 - Occasionally accompanied by loss of energy
- Lower respiratory allergies (asthma) in some allergic people characterized by:
 - Shortness of breath and Wheezing

These symptoms commonly occur absent mold exposure. They have many other causes, including viruses, pollens, dust, dust mites, cat and dog dander, as well.

General Causation and Wet-Damp Indoor Spaces

A number of studies have linked symptoms to either wetness-dampness in indoor spaces or to odors of mold indoors. In those studies, the matter of causality is, however, far from settled. Studies of the indoor environment and health are plagued by major methodological problems. To begin with wetness and dampness are generally self-reported by the study group and never verified. Those self-reports of dampness may range from a small spot of water under a sink to a major flood. Those hardly lead to similar potential exposures. Complaints, too, are rarely verified, but are rather self-reported. The characterization of a moldy odor is highly variable and subjective. Finally, the majority of studies are populated by symptoms or disorders that are never validated by reviews of medical records. As a result, the current belief among some that wet-



damp spaces cause symptoms absent an allergic basis is the subject of extensive study, but remains well short of settled.

The complexity of this issue is magnified by the many dozens of agents that have been associated with damp indoor spaces and to non-wet spaces as well, ranging from endotoxins, to mycotoxins, to glucans to bacteria. For example a study by Hicks et al (2005) found extensive mold in carpets of homes that had never been water damaged. None of the numerous identified agents found in homes has been specifically connected to any claimed health effects and studies of them individually often find no such connection.

It has also been hypothesized that people who have symptoms are more likely to report wetness and dampness in their homes. In other words, the symptoms, rather than being caused by wetness, may actually be reason for an over-reporting of dampness.

Lastly, there also exists a body of research which has linked early childhood exposures to microbiological agents in the indoor environment to protection from allergy, asthma and respiratory disorders. This has led to the "hygiene hypothesis," arguing that some exposures are salutary rather than harmful.

Thus, whether wet/damp indoor spaces produce symptoms or illnesses, other than by allergy, is argued, but far from proven. It is a complex and unsettled issue.

For the most part, the known and accepted health effects of molds and mold toxins, as well as other agents associated with wet-damp indoor spaces, are well-studied and widely published. Mold occurs naturally both indoors and outdoors. Mold spores occur in far greater concentration in the out-of-doors, but are transported indoors via mechanical ventilation, movement through open doors and windows, transport on materials brought into a building, or carried on humans, plants and animals. Some indoor surfaces can support the growth of mold. These materials include paper backing on wallboard, some types of ceiling tiles, wood, carpeting, fabrics and insulation materials made of cellulose. Indoor plants likewise are a source of mold. An ongoing water source is also necessary.

The clinical effects associated with mold/fungi exposure can be classified into three broad categories: allergic effects, infectious effects and potential toxicological effects. The latter, at issue here, are best known to occur following certain unusual and extreme exposure circumstances, not commonly related indoor air.



The allergic effects are primarily evident as respiratory allergies, such as hay fever or upper respiratory allergies, allergic rhinitis, asthma and, very rarely, hypersensitivity pneumonitis and allergic bronchopulmonary aspergillosis. Mold-related allergic effects are generally mild and occur only when actual exposure occurs. They are equally likely to result from exposure to molds out-of-doors, as well as from many other aero-allergens: pollen, grasses, dog and cat dander, feathers, dust mites, trees, weeds, house dust. Allergic responses occur to mold only in allergic people, who make up approximately 6-8% of the US population. Ms. Beckemeyer had some positive allergy tests to certain molds and to other agents as well.

Certain infectious mold disorders occur in immunocompromised individuals: for example, persons taking immunosuppressant medications, or those with immune suppressant diseases. In these vulnerable individuals, mold exposure may lead to infection in the lungs, sinuses, or even generalized infections throughout the body. These infections have not been seen in generally healthy individuals exposed to mold in the indoor ambient air. The scientific and medical literature also documents infections developing from exposure to outdoor molds endemic to specific areas of the country. Such infections have almost never been associated with indoor exposures.

The third category of adverse health effects associated with mold is that of mold toxicity. That seems to be what is claimed in this case. Several of Ms. Beckemeyer's physicians seem to believe that mycotoxins or other ill-defined "toxins" are responsible for her symptoms. One of the claimant's experts, Dr. McMahon, believes that various ill-defined agents in wet-damp indoor spaces produce some sort of toxicological effect which leads to an immunological disorder. He shares that belief with a small cadre of fringe practitioners whose methodology and self-described "disease" is not recognized or generally-accepted. They call this disorder chronic inflammatory response syndrome (CIRS or SIRS), a "disease" known only to them, not generally-accepted and not recognized by the American Academy of Allergy Asthma and Immunology (AAAAI), even though Dr. McMahon and likeminded practitioners claim this is an "immunologic disorder."

Many molds are capable of producing mycotoxins under specific circumstances of moisture, temperature and nutrition. Some mycotoxins can cause adverse health effects in humans. Such effects include adverse impacts on a variety of organs and metabolic processes, for example, aflatoxin affects the liver when ingested (not inhaled) in



sufficient amounts. Popular perceptions notwithstanding, the literature does not provide valid or accepted scientific support for the proposition that toxic effects associated with mold exposure are caused by the kind of low-dose inhalational exposures potentially encountered in indoor air. The studies which I shall describe elucidate the near impossibility of such effects.

No “CIRS” post Hurricane Katrina

If wet/damp indoor spaces produced the disorders claimed in this case, it would stand to reason that such disorders would have been found in homes following hurricane Katrina. Post Katrina, despite being extremely wet and moldy, exposures in these flooded homes did not lead to illnesses. The Centers for Disease Control (CDC) evaluated homes in New Orleans after Hurricanes Katrina and Rita. The authors of that evaluation found no evidence of adverse health effects associated with those exposures (Barbeau et al, 2010)

Mycotoxins

Specific mold species produce specific mycotoxins under specific conditions. The mere presence of such a mold is not evidence that it was producing any mycotoxin. Potentially toxigenic species of mold, including various species of the genera *Aspergillus*, *Penicillium* and *Stachybotrys*, do not always produce mycotoxins. Certain growth conditions are required and, in the case of *Stachybotrys*, different species produce entirely different mycotoxins ranging from nontoxic to humans to potentially-harmful at high doses. Some of the plaintiff’s causation experts in this case have assumed, with no support, that mycotoxins or other biotoxins were present in the car. None was actually measured. Moreover, as I will discuss later, airborne mycotoxins do not contribute to the body’s mycotoxin burden. On the other hand foodborne mycotoxins do.

In order for a toxic effect to occur, the toxin must be present, a route of exposure must be available and a sufficient dose must be received to produce the toxic effect. Mycotoxins are not volatile, nor are they cumulative (Bush 2006). An immutable principle of toxicology is that toxicity depends upon the dose of the toxic agent which enters the body. Thus, botulinum toxin is lethal in rather small quantities if eaten by people, but it is injected safely in millions (as Botox) to treat wrinkled skin. By diluting it a million-fold, Botox is converted from highly toxic to quite safe. The same is true for mycotoxins. As is discussed below, the calculated doses required for both acute and



chronic exposures to result in adverse health effects in humans are so high that it is essentially impossible for exposure in a home, let alone a car, to ever lead to a toxic adverse human health effect. The American College of Occupational and Environmental Medicine (2002 and 2011), the National Academies of Sciences' Institute of Medicine (2004), the American Academy of Asthma, Allergy and Immunology (2006) and the World Health Organization (2009) all concur that the scientific and medical evidence does not support the contention that mycotoxin-related disease (mycotoxicosis) occurs via inhalation of these agents in indoor environments. The current fact sheet of the US CDC concurs with that assessment: <http://www.cdc.gov/mold/stachy.htm> More specifically and pertinent to this case, none of these recognized organizations find any support for the proposition that either mycotoxins or other agents associated with damp indoor spaces, can produce either the range of symptoms alleged here or the "disease" "CIRS" made up by Dr. Shoemaker and adopted by Dr. McMahon.

Direct toxicological calculations show quite clearly that it is almost impossible for there to be enough mycotoxins in indoor air to produce toxicity (Kelman et al, 2004). They calculated that 200,000 spores/m³ of mold spores chock full of the most toxic of mycotoxins would be the necessary airborne concentration to produce toxicity. All levels of molds sampled in the car, in air and dust, were no different from common background levels found in non-water damaged homes. Only a fraction of those were of the type which the plaintiff's experts in this case might contend produce mycotoxins. In other words, even if these spores did contain those mycotoxins (a proposition which is speculative and unproven) the amount of those is thousands of times lower than the lowest level which is potentially toxic to human beings.

Toxic doses of mycotoxins can be taken into the body, primarily through ingestion of heavily-contaminated foods. This has been primarily an issue in farm animals which have eaten contaminated hay or grains. When a toxic dose of mold/mycotoxin is taken into the body, as might follow ingestion of heavily contaminated spoiled food, for example, a specific pattern of illness, well-documented in the literature, arises which is specific for given mycotoxins (Kuhn et al, 2003). That foods, not air, are the source of mycotoxins in our bodies was illustrated in two recent studies. In one, molds were found in homes which, in the laboratory, produced mycotoxins. However, in the homes in which those molds were found, the mycotoxins were not found (Jezak et al 2016). In another study, workers in grain grinding mills where airborne mycotoxin levels are quite high, had the same urinary mycotoxin levels as a control group whose exposure was purely dietary



(Follman et al 2016). Mycotoxins in our bodies and found in our urine come from food, not from air.

Fung and Hughson (2003) conducted a review of 416 studies in which no scientifically-sound evidence of a causal relationship between inhaled indoor mold toxins and any adverse health impact was found. This review also included, without specific comment, all other substances associated with wet/damp indoor spaces since that was setting which gave rise to these studies. Mold toxin risk assessment was reported by the American College of Occupational Environmental Medicine (ACOEM) in its 2002 consensus document which was reaffirmed in 2011. This well-respected, relevant medical organization determined that, even if some mycotoxins are produced and become airborne and accessible for human exposure, the amount and duration of exposure shown to cause adverse health effects was extremely high: far higher than ever occurs with indoor contamination.

In 2004, the Institute of Medicine (IOM), National Academies of Sciences, thoroughly reviewed the available medical and scientific literature to determine whether there was any association between exposure to damp indoor environments where mold and other agents grow and a variety of symptoms, conditions and diseases. The IOM found no evidence of an *association* (not causation) between exposure to damp indoor environments and the majority of symptoms alleged in this claimant.

Similarly, in its 2009 *Guidelines for Indoor Air Quality: Dampness and Mould*, the World Health Organization specifically noted that, “no study has shown that people living in damp buildings who complain of nervous system symptoms are exposed to effective levels of mycotoxins.” WHO concluded, “The evidence that [mycotoxins] play a role in health problems related to indoor air is extremely weak.” Further “toxicological studies” were called for, in order to “clarify and identify causative compounds,” but the authors of the *Guidelines* warned that, “The dosage must be considered in interpreting the findings and attempting extrapolation to the range of human exposures indoors.” To date, there exist no such studies.

In a more recent report, the German Society of Hygiene, Environmental Medicine and Preventive Medicine reached similar conclusions (Hurras et al 2017).



For all of these reasons, the notion that any mycotoxin effects arise from mold in homes or from a limited period of driving in a car is erroneous. A small group of zealous advocates, a few of whom are involved in this case, as well as the plaintiff, may believe otherwise, but their views are scientifically-unsupported, out of the mainstream and not generally-accepted.

Specific causation in an individual

Assuming that the general causation requirements can be met, which is not the case in the Beckemeyer matter, specific causal elements would need to be evaluated and ruled in or out.

The causation methodology used to establish specific causation, that is, causation in an individual, is widely-accepted in the literature (Gots 1986, Gots 1993, Hackney 1979, Evans 1976, Irey 1976, Schwartz 1995, Tarcher 1992, Marley 1991, Buffler 1995, Rom 1992, Black 1990, Black 1993, Sullivan 1992, NRC 1992, Brennan 1987, Guezalian 2005, Weiner et al, 2012). Other accepted methodologies differ in detail, but not in basic principle. The elements of the causation methodology used to establish specific causation include the following:

How was the diagnosis made?

Does the patient have a recognizable disease?

Are we dealing with symptoms alone or with objective disorders?

Have other causes been properly considered and ruled out? Has the exposure been confirmed?

Was the dosage sufficient considering the concentration and duration to produce the condition(s)?

Was the clinical pattern what one would expect from that causal agent?

Were the temporal relationship and/or latency periods appropriate?

All of these questions and their answers are highly individual and must be addressed symptom (or disorder) by symptom (or disorder).



The discussion of those causal elements in this case will follow.

Specific Causation in This Matter

None of the specific causation requirements are met in this case, as the detailed review above demonstrates. In fact, all of them are specifically refuted.

How was the diagnosis made? And

Does the patient have a recognizable disease?

The primary diagnoses in this case made by standard practitioners were recognized respiratory disorders—allergies and infections. Others were major anxiety disorders. The subsequent diagnoses, and the subject of this claim, CIRS, are not recognized diseases, but the creation of non-traditional practitioners and neither scientifically-known nor generally-accepted.

Are we dealing with symptoms alone or with objective disorders?

For some of Ms. Beckemeyer's symptom complaints, specific clinical findings are apparent in the records. These exclusively involve the respiratory tract. She allegedly has certain laboratory abnormalities, according to several of her non-mainstream practitioners. Those will be discussed later.

Have other causes been properly considered and ruled out? Has the exposure been confirmed?

There was no exposure to mold in this car. All levels were low and/or typical everyday background levels. There was also no wetness established. Thus, no exposure has been confirmed.

Other causes of her symptoms and findings have been identified including allergies, infections, major anxiety and depressive disorders. She is also clearly persistently anxious which explains many of her symptoms.

Was the dosage sufficient considering the concentration and duration to produce the condition(s)?



As a board certified toxicologist, I can unequivocally state that there were no toxicological agents in this car capable of producing acute or chronic illnesses.

Was the clinical pattern what one would expect from that causal agent?

Ms. Beckemeyer's clinical pattern of symptoms, objective findings and response to treatment supported the allergic and infectious conditions diagnosed by her standard practitioners. This pattern is not consistent with the car having been the cause.

That some mold-related or other unspecified "toxin" related to the car was causal of this claimant's made-up "condition," CIRS, is completely unsupportable. This condition has no specific clinical pattern, no objective findings or standard clinical testing; it has a variable array of wide-ranging, subjective symptoms which can occur in virtually any organ system in a pattern defined solely by the patient herself.

Were the temporal relationship and/or latency periods appropriate?

The timing of symptoms and the location of their occurrences also speaks clearly against the car as responsible. While Ms. Beckemeyer later claimed that she was sick from the moment she got the car, contemporaneous medical records do not support that. She had acute infectious respiratory conditions for short periods during her three months with the car. She also had allergic symptoms which were ascribed to non-car associated exposures. She later, after disposing of the car, developed most of her symptoms which persisted and, in some instances, intensified. Thus, there is a clear temporal disconnect between the car and her primary complaints.

Very importantly, she had longstanding prior sinus and respiratory disorders as well as major anxiety disorders. Responsible for many of her symptoms, these long predated the car issue.

The bottom line of this causation analysis is that there is clear evidence against specific causation. The car played no role, except a perceived one, in Ms. Beckemeyer's illnesses or symptoms.



The role of perceptions of hazards in symptom development and reporting. An alternate specific causal factor

An important specific causal factor in many exposure matters relates to fear or worry by the individual. Symptoms are frequently over reported when people believe their health has been threatened. A review of the scientific literature regarding self-reported symptoms indicates that these can be unreliable when perceived hazards exist as a basis for confirming health problems. Over the years, numerous authors have studied and reported upon the unreliability of self-reported symptoms, particularly following perceived toxic exposures. (Gots et al, 1992, Hopwood et al, 1988, Lees-Haley et al, 1992, Kaye et al, 1994, Lipscomb et al, 1991, Lipscomb et al, 1992, Logue et al, 1986, Pennebaker et al, 1994, Roht et al, 1985). The most important reason is the well-known phenomenon of reporting bias. (Lipscomb et al, 1991, Logue et al, 1986, Pennebaker, et al, 1994, Last et al, 1992, Hennekens et al, 1987, Pennebaker et al, 1983) Reporting bias, a standard epidemiological term, is not a pejorative as it is in common usage. Rather, it reflects the normal human tendency to connect physical phenomena with unrelated causes, particularly when the perceived cause is viewed as a health threat. Extensive media coverage of hazards (such as waste sites, chemical spills, or nuclear accidents) affects perception of risk. (Aakko 2004) The “hazard du jour” phenomenon begins with media reports of a link between a health effect and a consumer product or environmental factor. (Kabat 2008). Mold and mold toxins is one of those current popularly perceived hazards.

Studies of health complaints of residents living near waste sites have explored the relationship between alleged low level chemical exposures and verification of the actual diseases. These have reported negative results in most instances. (Dunne et al, 1990, Naker et al, 1988, Hertzman et al, 1987, Janerich et al, 1981) When questioned, individuals living near a waste site almost always produced more complaints than individuals living in an area without such a waste site. Individuals concerned about the quality of indoor or workplace air tend to report a wide range of health complaints. These increased complaints, unassociated with verified, actual disease, emphasize the intensity of the belief about a toxic risk held by the reporting individuals. When individuals perceive a risk from something over which they have little or no control they experience a higher level of threat and fear. (Dunne et al, 1990, Baker et al, 1988).



One study illustrates quite clearly the unreliability of self-reporting. It involved a New Jersey community whose water supply was reported to be contaminated. When “unexposed” and “exposed” populations in Somerset, New Jersey, were questioned, the symptom responses of the group that believed it was exposed were substantially higher than that of the group residing in another part of New Jersey. Evidence obtained after the study revealed that the “exposed” group actually had not been exposed. Their water was not found to be contaminated, although they had believed it was. Clearly the symptom reporting on the part of these residents was directly related to the perception that exposure had occurred (NJDOH 1983). Numerous other studies in the published literature have reported precisely the same findings (Pennebaker 1994). When perceptions of hazards increase, symptom reporting increases, whether or not hazards actually exist.

At times, physicians can play a role in initiating and maintaining patients’ symptoms and illness beliefs. Kellner (1990) made the observation that “in many somaticizing and hypochondriacal patients, iatrogenic (physician-induced) reinforcement played a part,” in encouraging and maintaining the patients’ symptoms or believed illnesses. It has been pointed out that with the increased awareness of chemicals in the environment and media attention to indoor and outdoor air issues, as well as to the hazards of chemicals, there is a greater tendency for psychological factors to aggravate, and even cause, a multiplicity of symptoms. Physicians, too, share the public’s awareness about chemicals and are exposed to the same media information about alleged hazards. Even when physical findings are few or absent, physicians, at times, will diagnose illness and attribute it to the exposure reported by the patient. By misattributing symptoms to a cause or diagnosis of questionable validity, a physician can encourage and reinforce a patient’s symptoms and belief in illness.

Nontraditional practitioners and an even larger number of physicians and health care providers, who share some of their beliefs and practices, routinely diagnose environmental illnesses. Black (1996) published a paper entitled *Iatrogenic* [sic physician-caused] *Multiple Chemical Sensitivities*, in which he described this phenomenon. He noted that iatrogenic reinforcement can play a critical role in initiating and maintaining illness belief. In the case of mold and mold toxins, I have mentioned the role that Dr. Shoemaker and his followers have played. Dr. Shoemaker will be discussed below in more detail.



Finally, it is important to determine whether symptoms are the result of an emotional response to a perceived chemical toxicity or to an abnormal physiological interaction between chemical agents and organ systems. It is not possible from symptoms alone to tell which is the case, since either source can produce identical symptoms (Gots 1996). The recognition of the origin of complaints, acknowledgement of reporting bias in patients' reporting of symptoms, as well as the public's fear of environmental hazards, all contribute to symptom reporting, as can true pathologic health effects produced by environmental agents. Mood, feelings, stressors, fears and worries can all have profound impacts upon symptom development and even disease. The need to identify the primary or predominant causal factors of the symptoms, whether emotional or organic, is essential. Making such a distinction is neither trivial nor unnecessary. A brain tumor would not be treated with psychotropic drugs any more than stress-related headache would be treated with neurosurgery, even though both cause headaches. Both stage-fright and an abnormal electrical pathway in the heart can cause palpitations; each is treated differently. The critical distinction between an organic (i.e., toxicogenic) and psychogenic disorder is essential to make and can be made through proper testing (Gots 1996).

The Role of Stress in Immunological parameters

Perceived exposure to a hazard is a stressor which, as noted, can and does produce symptoms. It also can produce measurable immunological changes. In other words, laboratory values are affected by stressors. Numerous studies over the years have addressed this as they have studied the role of stressors in the production of a wide variety of illnesses—susceptibility to infection, heart disease, cancers and many others. One of the largest studies of this phenomenon, performed a meta-analysis of 300 scientific articles on this subject (Segerstrom et al 2006). Chronic stressors (specifically, unemployment was one) had the most marked effect on measured immunologic parameters. Ms. Beckemeyer was unemployed and was told she was poisoned; she reported to Dr. Shatz her fear about her future employability. Those are clearly significant stressors. In addition to Segerstrom, others have discussed the mechanism by which stress affects immunological parameters. Among them is an article entitled *Psychoimmunology examined* (Thornton et al 2006). There is, in fact, an entire field of study known as “Psychoimmunology.”



CLAIMANT'S EXPERTS' UNCONVENTIONAL CAUSAL THEORIES IN THIS MATTER

A number of medical “experts” are supporting Ms. Beckemeyer’s beliefs in this matter or are mentioned. They are among a relatively small number of practitioners, outside the mainstream, whose diagnoses, attributions and treatments are neither generally accepted, nor scientifically supportable. In this case, they include Drs. Cleveland, Blatman, Pretorius, Huber, McMahon and Shoemaker. They have diagnosed SIRS or CIRS or toxic mold exposure or biotoxin illness. SIRS is actually a condition referring to terminally ill patients, most often in ICU, who have multi-system failure. CIRS is a diagnosis made up by Dr. Shoemaker and adopted by Dr. McMahon, but not generally accepted. They believe that CIRS is a “biotoxic” illness.

To elucidate where this term “biotoxic illness,” “toxic mold exposure” and CIRS come from, I shall discuss two of the involved physicians—Drs. McMahon and Shoemaker—who are central players in this non-accepted disorder.

The claimant’s expert, Dr. McMahon, is relying upon several “evidentiary” approaches to support his causal allegation. All are wrong and defy accepted medical and toxicological reasoning.

The first is the general causation belief which I have discussed above. That is, that mycotoxins or other factors coming from indoor environmental air are capable of leading to the manifestations alleged and the strange self-named, unrecognized diseases CIRS. That is untrue, has never been shown scientifically and is not generally-accepted. The US CDC in its current online report agrees (<http://www.cdc.gov/mold/stachy>), as do all of the other consensus groups mentioned above.

The second is that the exposure and consequent dose of such toxins allegedly received by Ms. Beckemeyer of mycotoxins or anything else was sufficient to cause the complaints. To “establish” this, the supporting practitioner is relying on several lines of self-developed and self-serving “evidence.” The first is the unproven and likely untrue assertion that “toxins” or other inflammation-inducing agents were present and in sufficient quantities to exert effects. The second, is that those mold toxins, if mold toxins are at issue, came from the air of the car interior. The third is that the complained of disorders are consistent with toxicity associated with indoor environments. Finally, the fourth is that various odd laboratory tests which he orders, supports his diagnosis and his causal attribution.



If Ms. Beckemeyer were exposed to a mycotoxin, as she and some of her experts seem to believe, and even if they were to enter the body, they are rapidly eliminated. Any mycotoxins theoretically entering her body arising from the car would have been essentially gone by December 2016. However, everyone has levels of mycotoxins in their bodies, arising from dietary sources

Like all other foreign substances, such as medications and most other toxins, mycotoxins are broken down and eliminated by the body. Mycotoxins are metabolized in the liver and excreted by the kidney in short order. Numerous scientific studies have examined the time that it takes for mycotoxins to be eliminated. In toxicology and pharmacologically, this rate of elimination is generally called “half-life.” This is the time that is required for one half of the absorbed dose to be eliminated. For the most part, the half-lives of studied mycotoxins occur in minutes to a few hours. Thus, they do not persist for weeks, months or years after exposure cease and, as a result, are not, capable of producing increasing or persistent symptoms or causing new symptoms to arise.

The presence of toxin-producing molds and of mycotoxins or any other “toxic substance” in the car at issue has not been shown. There were no measurements performed. Furthermore, the total mold levels found in the car which is the subject of this case, were not unusually high or indicative of significant water intrusion and active mold growth. They were common everyday exposure levels. Even if the mold spores found were full of mycotoxins, thousands of times more than the largest amount found would have to have entered the body to produce toxicity. As recent studies have shown, airborne mold or mold toxins do not contribute to internal levels of mold toxins. Those come from dietary sources (Follman et al, 2016, Jezak et al, 2016).

Dr. McMahon and a couple of Ms. Beckemeyer’s treating physicians are basically followers of a Maryland physician, Ritchie Shoemaker, who popularized, among a fringe medical group, the diagnoses and testing which they follow. The fact is that these individuals belong to a small cadre of alternative practitioners who have promoted false notions about indoor environments and all manners of adverse health consequences associated with them. Their beliefs and practices are neither generally-accepted nor scientifically-known.



Dr. Shoemaker's practices came under fire by the Maryland Board of Medical Examiners in 2013, when he was reprimanded and put on probation for failing to meet proper standards of care. At that point, he discontinued his clinical practice. However, he now purports to provide special certification to physicians who follow his diagnostic and therapeutic methods. Dr. McMahon, an expert in this case, and a pediatrician by training, touts his mold certification by Shoemaker as his primary credential in this arena. He adds to that the assertion that he has treated thousands of such patients, solidifying his self-proclaimed expertise and diminishing that of anyone who does not have such clinical experience. Medical history tells us that even large numbers of diagnostic and treatment experiences do not necessarily imply knowledge or wisdom. No doubt the colonial physician who bled George Washington would also have trumpeted his experience with thousands of such treatments.

Drs. Cleveland and Blatman may not have been formal students of Dr. Shoemaker, but they used his earlier diagnostic terminology of SIRS. They used the Shoemaker "protocol" to treat it in Ms. Beckemeyer.

Since approximately 2008, Dr. Shoemaker has called his novel disorder Chronic Inflammatory Response Syndrome (CIRS), a condition which is neither generally accepted nor known to exist. Ms. Beckemeyer, Drs. Cleveland, Blatman and McMahon use that term or a variation of it, SIRS. Dr. Shoemaker and his followers further claim that this disorder results from exposure to a variety of "biotoxins" associated with wet/damp indoor spaces. This theory is self-generated, unproven, based on poor causation and scientific methodologies, and resides in questionable data. It is not generally-accepted.

Dr. Shoemaker originally called the condition Sick Building Syndrome (SBS) and Chronic Biotoxin-Associated Illness, before he renamed it "Chronic Inflammatory Response Syndrome." Initially, he attributed this entity to a mycotoxin cause. In 2002, he consistently attributed symptoms to "toxin-forming species of fungi" and their mycotoxins. In his book *Mold Warriors* (2005), Dr. Shoemaker wrote similarly about the cause of illness in the cases he discussed. He highlighted water damage and mold growth and that certain molds make toxins, "our studies show repeatedly: mold makes people sick." (p. 332-333). He has subsequently disavowed the role of mycotoxins, believing instead that, "exposure to mycotoxins is a 'relatively insignificant factor in the systemic inflammatory response these people get'." (See Deposition testimony in *Anderson et al*



vs. *The Ritz Carlton Hotel Co. et al.*, DC Superior Court Civil Div. CA No. 05ca (0001130).)

Certain of the diagnostic tests performed on Ms. Beckemeyer, such as VCS, MSH, TGF, and C4a, even the so-called HLA-genotype allegedly associated with environmental exposure leading to symptoms and a dozen others are neither scientifically-known nor accepted as showing what they are claimed to demonstrate. They are not described in the recognized and accepted medical and scientific literature for this purpose. Many of these tests have little clinical application at all, but are unique to certain specialized research laboratories, or to either specific other disease states or to other illnesses. These tests taken together are not delineated in textbooks or published as part of evidence-based clinical guidelines used by the general medical community. They are the sole province of this fringe medical group who call themselves “environmental physicians,” or, now, “certified mold physicians.”

Like the changes in name and causes of his novel illness, Dr. Shoemaker’s diagnostic tests and the parameters measured by them have changed, as well. He admitted in testimony that, “the normal set of labs that I use also evolves over time. If you look at what I did in 2003, it contains many of the same elements I use in 2008. Some have come and gone out of that list from 2003, other new ones have come and gone and other new ones have come and stayed.” (Testimony in *Anderson et al.*, 22 May 2008, p. 28, l. 20-25). His diagnostic tests, then, are a moving target: there is no way that they could constitute a reliable, validated test battery which has become generally-accepted. With both the parameters being tested and the tests themselves changing or “evolving,” it is little wonder that the information upon which he and his followers base their conclusions is not found in journals that are generally available to and accepted by the medical community. They are, rather, their personal, idiosyncratic characterizations and creations, neither reliable nor validated for the purpose for which they are employed.

Many of the tests are discussed in Dr. Shoemaker’s articles describing his “studies” and theoretical “mechanisms” for his personally-defined diseases of “sick building syndrome” and “chronic biotoxin-associated illness from exposure to water-damaged buildings.” Those were published in *Neurotoxicology and Teratology* (Shoemaker et al, 2005; Shoemaker et al, 2006). He has recently published another article dealing with “neuroquant testing” in that same journal (Shoemaker et al, 2014). Dr. Shoemaker has asserted that all articles were peer-reviewed. There is, however, no evidence that these



articles are peer reviewed or, if they are, that they are reviewed with serious rigor. Dr. McMahon, has recently published related articles in a journal called *Medical Research Archives*: a journal with an impact factor of about 1, meaning that it is rarely read or cited.

The journals in which these studies were published, *Neurotoxicology and Teratology*, and *Medical Research Archives* are not among those journals listed by the International Committee of Medical Journal Editors (ICMJE) as following its “Uniform Requirements of Manuscripts Submitted to Biomedical Journals.” Journals which have agreed to follow its peer-review standards, as well as the other requirements delineated, are listed by the ICMJE. There are at least 1000 of those, but the two noted above are not included.

I serve as a peer reviewer of a standard, recognized journal. Having read Dr. Shoemaker’s and Dr. McMahon’s papers, I can state that they would not pass generally-accepted requirements of a peer-reviewed journal.

In assessing whether a physician or scientist used reliable scientific methodology to reach his conclusions, it also is important to examine the known or potential rate of error of a particular theory. For example, if a scientist placed various qualifications on his conclusions, then the known rate of error is potentially very high. The rate of error cannot even be assessed for a theory that has not been tested. Dr. Shoemaker’s and Dr. McMahon’s theories remain in the unreplicated, unreliable category. Their alleged scientific methodology in reaching their conclusions about this “CIRS” condition have, therefore, not been shown.

Another important factor in assessing the scientific reliability of a theory is whether the theory has gained widespread acceptance among scientists in that particular field. General acceptance indicates that other scientists agree that a theory is based upon reliable scientific methodology, has been replicated and has scientific validity. If, over time, a scientist’s opinion has gained little or no support within a particular field of science, it is appropriate to question whether the opinion is supported by reliable scientific methodology. The term CIRS as a disease entity has been used by Dr. Shoemaker since at least 2008. Dr. McMahon says that the condition was first described in 1997 (McMahon 2017). Since it is, according to them, a widespread immunological disorder (Dr. McMahon now claims 7% of people have CIRS), one would think that if it were proven, by now it would be widely-accepted. It is not. It is not recognized, for example, by the American Association of Allergy Asthma and Immunology, the major



immunology body in this country. It also has no ICD-10 number, that is, no number in the International Classification of Diseases, version 10. Dr. McMahon dismisses this, suggesting that such numbers are only for “insurance purposes.” Actually, the disease classification numbers are established by the World Health Organization to provide uniform nomenclature for study purposes, so that physicians and scientists worldwide are speaking about the same entity when they do studies. There is no ICD-10 number for CIRS.

Thus, the notion that there exists a “biotoxic illness” or “SIRS/CIRS” due to the inhalation of mold toxins and other things in the indoor environment is not one which is generally-accepted or scientific knowledge in the medical community. The current state-of-the science is discussed by organizations noted earlier, including the US CDC (<http://www.cdc.gov/mold/stachy>)

Dr. McMahon, an expert in this matter, claims proudly to be a disciple of Dr. Ritchie Shoemaker. He diagnoses the Shoemaker disease, now named Chronic Inflammatory Response Syndrome (CIRS), which he believes arises from indoor exposures and attacks every organ in the body. He diagnoses this disease through a set of thirty or more disconnected symptoms and a bizarre array of laboratory tests only performed in this combination by Dr. Shoemaker’s zealous followers. This disorder of Dr. Shoemaker’s is trumpeted in papers that he has written, most published in obscure, non-mainstream journals or conference proceedings. At this point, approximately eight of his papers have been in the literature for at least ten or more years old, yet his revolutionary revelation has never found its way into mainstream medical or scientific journals.

It has also never made it into any of the most authoritative consensus documents, such as the World Health Organization (WHO) and the Institute of Medicine, National Academy of Sciences (IOM/NAS) publications on wet damp indoor spaces. Although many of Dr. Shoemaker’s papers were available by the time the WHO document was published, there are no references within it to Shoemaker or to this “CIRS disease.” Dr. McMahon cites extensively from the WHO document and from another, the Government Accountability Office (GAO) document of 2008, which he claims supports him and Dr. Shoemaker. However, neither document mentions any of Dr. Shoemaker’s “groundbreaking research,” cites any of his papers, or contains any discussion whatsoever of this disease “CIRS,” which is the centerpiece of Dr. McMahon’s long report in the Beckemeyer matter. Dr. McMahon’s notions, methodologies and diagnosis,



a clone of the Shoemaker beliefs, are neither generally-accepted, nor scientific knowledge, nor reliable.

Dr. McMahon's report employs all of the well-recognized rhetorical flourishes of the self-promoter in any area of endeavor. It touts his and Dr. Shoemaker's thousands of patients experience, denounces his enemies as "naysayers" and ill-informed troglodytes who are in the pocket of special interests and uses all manners of tangential "evidence" to support his position. Boasting of thousands of patients is the technique of the practitioner who is trying hard to convince the world of his miraculous breakthrough. If his notions were generally-accepted, however, would that be necessary? One would not expect a diabetologist to crow about his 3000 diabetic patients or a cardiologist to brag about the 5000 patients he has treated with Lipitor. Considering the potential number of wet/damp buildings, homes and cars and the frequency of the genetic susceptibility that Dr. McMahon claims, hundreds of thousands of people should be suffering from this devastating and debilitating chronic disease and the medical community should have long recognized this critically important new disorder. If innumerable individuals like Ms. Beckemeyer were falling victim to illnesses from this CIRS, the medical community at large would have embraced, or at least considered, that possibility. Are all conspiring with homeowner's or auto insurers or landlords? Dr. Shoemaker has been pushing his belief and writing about it for nearly twenty years. The simple and obvious answer is that Drs. Shoemaker's and McMahon's "disorder" and its so-called confirmatory diagnostic testing are scientifically bereft.

Nowhere in the GAO report which Dr. McMahon cites does it say that we now know of an inflammatory disease described by Dr. Ritchie Shoemaker known as "CIRS" or "biotoxin illness" or "mold illness" or any other, which produces widespread injury because of a chronically over-reactive innate immune system. In fact, that 61 page document which Dr. McMahon relies upon extensively contains not one mention of CIRS, of him or of Dr. Shoemaker.

The WHO report discusses many possible mechanisms which might account for symptoms associated with exposures to WDB's. Within this hypothesized group are included possible immunological mechanisms and inflammatory mechanisms. Dr. McMahon and Dr. Shoemaker, have elevated these preliminary considerations by proclaiming them established fact and claiming that this validates their idiosyncratic "disease" "CIRS." In fact, WHO considers many possible toxicological mechanisms



including: Immunostimulation and Immunoglobulin E-mediated allergies; Cytotoxicity and Immunosuppression; Autoimmunity; and Irritation (pp84-89). These postulated mechanisms underlie current theories and animal research, but are far from accepted, established answers to the human health effect questions. In the summary of that section, the WHO report says: “The mechanism by which non-infectious microbial exposures contribute to adverse health effects associated with indoor air dampness and mould are largely unknown.” (p 90)

By contrast, Dr. McMahon would have us believe that the mechanism is clear and resolved. It is his disease “CIRS,” which explains it all, yet that term was not mentioned once in the WHO report. Thus, Dr. McMahon’s note that “[i]mmunologic aspects’ of illnesses and symptoms associated with WDB are considered by WHO,” is true, but it is not true to say that they were “emphasized” to the point of being definitive, as Dr. McMahon has suggested. Furthermore, “immunologic mechanisms” is a very broad category. Suggesting that such a category is the equivalent of the Shoemaker/McMahon unrecognized disease “CIRS” is highly misleading and flat out wrong.

Dr. McMahon claims that this disease, so-called “CIRS,” is an immunological disorder. As noted earlier, it is not recognized by mainstream immunologists or the many thousand members of the American Academy of Allergy Asthma and Immunology (AAAAI), the official AMA recognized body of immunologists. Furthermore, one wonders why, if this disorder is an immune system disease, Dr. McMahon doesn’t refer Ms. Beckemeyer to an immunologist. He, after all, is a pediatrician.

A key to Dr. McMahon’s diagnosis of this CIRS is laboratory testing for a variety of parameters which he claims, when taken together, establish the diagnosis. To optimize the so-called “abnormals,” he creates his own normal values which, in some instances, differ from those of the laboratories. When Dr. Shoemaker was challenged years ago about his “normal” value for MSH which differed from the laboratory’s norm or reference range, he asserted that the low end of their reference range was “skewed by all of his patients’ ” abnormally low values. Dr. McMahon has done exactly the same thing in his Beckemeyer report. The fact is that both major commercial laboratories, Quest and LabCorp have an MSH reference range [range of normal values] that start at zero and, in the case of LabCorp, range from 0-40 and, in the case of Quest, range from 0-100. LabCorp is the lab Dr. McMahon used. He insists that the norm should be greater than 35; thus, Ms. Beckemeyer was abnormal at 5. In other words, he made up his own



normal to fit his own case definition. According to both these laboratories, her MSH was normal.

He did the same thing for the ADH determination which he says was abnormal at Ms Beckemeyer's level of 1. The laboratory which performed the test, LabCorp, reports its normal as 0-4.7. Dr. McMahon says the normal range is 1.3-8 making Ms. Beckemeyer abnormally low, according to him, but normal according to the testing laboratory.

Thus, at least two of these key tests that he relies upon were, according to him, abnormal. His assertion is contradicted by the reference standards of the laboratory which performed those tests and found them normal.

It is axiomatic in toxicology that the dose makes the poison. Dr. McMahon rejects this bedrock toxicological axiom. The reason is that he never has any idea of the nature or extent of his patients' exposures. Thus, whatever the exposure, it was, according to him, sufficient. So by Dr. McMahon's logic, the mycotoxins in our daily diet would have to be equally causal or, at least, could not be ruled out as the cause of the alleged disease in these individuals like Ms. Beckemeyer. Dr. McMahon's version of toxicology would make commonly-used Botox an invariably lethal substance since, in its undiluted form, botulinum toxin is an extremely potent and lethal agent.

In order to avoid evaluation and criticism of his CIRS disorder by toxicologists, Dr. McMahon claims, in his report, that it is not a toxicological disease, but an immunological one. However, in a paper which he wrote in 2017 and references in this report, he says: "In all patients (sic, CIRS patients) environmental exposures to biologically-produced toxins trigger innate immune cytokine overproduction..." In other words, he says in this paper, as he and Dr. Shoemaker have said many times, that their claimed "immune system disease" is the result of a toxic exposure.

In an attempt to enhance the respectability and acceptance of his "disease" "CIRS," Dr. McMahon claims that there exist 100,000 references on Systemic Inflammatory Response Syndrome. That disorder (SIRS) is indeed coded in the ICD-9 and ICD-10, as he notes. However, SIRS has nothing to do with his CIRS. SIRS is a code reserved for severe multiorgan-system disease and is used predominantly in patients in ICUs or trauma centers.



Dr. McMahon quotes the “Policyholders of America’s Research Committee Report on Diagnosis and Treatment of Chronic Inflammatory Response Syndrome caused by exposure to the interior environment of water-damaged buildings (2010),” as the most comprehensive paper on biotoxin-related illness to date. He claims that it shows that the world’s academic community recognizes this disease. It does nothing of the kind. It is a manifesto written by Drs. McMahon and Shoemaker, espousing the same positions promulgated in this report regarding Ms. Beckemeyer. The site “Policyholders of America” is devoted to “mold sufferers” and their supportive physicians. There is nothing academic about it.

Dr. McMahon, and Dr. Shoemaker before him, treat their “CIRS” patients with cholestyramine (CSM) and Welchol, drugs which are marketed to reduce cholesterol levels, but have largely been replaced by more effective agents such as statins. They use these allegedly to bind mycotoxins in the GI tract, but how supposedly-inhaled mycotoxins get to the GI tract awaiting binding by these drugs is unclear to me and to other toxicologists. Furthermore, Dr. McMahon makes a major issue about why ingested mycotoxins pose little risk because they are digested, broken down and rendered inactive in the GI tract. If that is so, why would he need to remove them with CSM or Welchol? Additionally, he states that mycotoxins are not the primary issue, but only one of many.

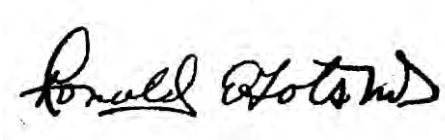
Despite that, he claims that his patients improve after being treated with this agent which allegedly eliminates mycotoxins. Lastly, as I pointed out earlier, any theoretically-present mycotoxins from the car at issue would have been long gone from the body by the time Ms. Beckemeyer received the agents in early 2017. The inconsistencies between the alleged efficacy of CSM and Welchol and Dr. McMahon’s various theories are enormous and irreconcilable.

For all of the above reasons, Dr. McMahon’s opinion in this case is far outside the mainstream of medicine. It is not generally-accepted and is scientifically unreliable.



CONCLUSIONS

Nothing in these records—medical or environmental testing—indicates a problem with this car or a disorder in Ms. Beckemeyer having anything to do with her car. Her ailments and symptoms have clear alternate explanations. There was no temporal connection between those and her use of the car. There was no in-car exposure to the alleged causal agents. Her primary experts have used unconventional, unaccepted methodologies and diagnoses to assert, incorrectly, a causal relationship. These opinions are expressed with scientific and toxicological certainty.



Ronald E Gots, MD, PhD, DABT

December 1, 2018



REFERENCES

Aakko E. Risk communication, risk perception and public health. *WMJ*. 2004;103:25-27

American College of Occupational and Environmental Medicine (ACOEM) 2002, "Adverse human health effects associated with molds in the indoor environment", *ACOEM Evidence-based statement: Position Statement/Guidelines*.

American College of Occupational and Environmental Medicine (ACOEM) 2011, "Adverse Human Health Effects Associated with Molds in the Indoor Environment," *ACOEM Position Statement*

Barbeau, DN, Grimsley, LF, White LE, El-Dahr JM and Lichtveld, M. 2010 " Mold Exposure and Health Effects Following Hurricanes Katrina and Rita," *Annual Rev. of Public Health* vol 31: 165-178.

Baker DB, Greenland S, Mendlein J, Harmon P. A health study of two communities near the Stringfellow Waste Disposal site. *Arch Environ Health*. 1988;43:325-334.

Baxter, D. M., Perkins, J. L., McGhee, C. R., & Seltzer, J. M. 2005, "A regional comparison of mold spore concentrations outdoors and inside "clean" and "mold contaminated" Southern California buildings", *J.Occup.Environ.Hyg.*, vol. 2, no. 1, pp. 8-18.

Benninger, M. S., Ferguson, B. J., Hadley, J. A., Hamilos, D. L., Jacobs, M., Kennedy, D. W., Lanza, D. C., Marple, B. F., Osguthorpe, J. D., Stankiewicz, J. A., Anon, J., Denneny, J., Emanuel, I., & Levine, H. 2003, "Adult chronic rhinosinusitis: definitions, diagnosis, epidemiology, and pathophysiology", *Otolaryngol.Head Neck Surg*, vol. 129, no. 3 Suppl, pp. S1-32.

Bischof W, Koch A, Gehring U, Fahlbusch B, Wichmann HE, Heinrich J. Predictors of high endotoxin concentrations in the settled dust of German homes. *Indoor Air*. 2002;12:2-9.



Black DW. Iatrogenic (physician-induced) hypochondriasis. Four patient examples of "chemical sensitivity". *Psychosomatics*. 1996;37:390-393.

Blanc PD, Eisner MD, Katz PP et al. Impact of the home indoor environment on adult asthma and rhinitis. *J Occup Environ Med*. 2005;47:362-372.

Black B, Warshaw MS, Hollander DH. Unraveling causation: back to the basics. *Toxics Law Reporter*. 1993;1061-1070.

Bush, R. K., Portnoy, J. M., Saxon, A., Terr, A. I., & Wood, R. A. 2006, "The medical effects of mold exposure, AAAAI Position Paper", *J Allergy Clin Immunol.*, vol. 117, no. 2, pp. 326-333.

Dunne MP, Burnett P, Lawton J, Raphael B. The health effects of chemical waste in an urban community. *Med J Aust*. 1990;152:592-597.

Environmental Health Hazard Evaluation Program, New Jersey Department of Health, and DHEHHEP. Health Survey of the Population Living Near the Price Landfill, Egg Harbor Township, Atlantic County. 1983.

Egner PA, Groopman JD, Wang JS, Kensler TW, Friesen MD. Quantification of aflatoxin-B1-N7-Guanine in human urine by high-performance liquid chromatography and isotope dilution tandem mass spectrometry. *Chem Res Toxicol*. 2006;19:1191-1195.

Follmann W, Ali N, Meinolf B, Degen GH, "Biomonitoring of Mycotoxins in Urine: Pilot Study in Mill Workers." *Journ. of Toxicol. and Environmtl Health*. 2016; vol 79, nos 22-23,pp 1015-1025.

Fazekas B, Tar A, Kovacs M. Ochratoxin a content of urine samples of healthy humans in Hungary. *Acta Vet Hung*. 2005;53:35-44.

Fung, F. & Hughson, W. G. 2003, "Health effects of indoor fungal bioaerosol exposure", *Appl Occup Environ Hyg*, vol. 18, no. 7, pp. 535-544.

Gots, R. E. 1986, "The science of medical causation: its application in toxic tort litigation", *For the Defense*, vol. 1986, no. 4, pp. 18-26.



Gots RE, Gots BA, Spencer J. Proving causes of illness in environmental toxicology: 'Sick buildings' as an example. *Fresenius Envir Bull.* 1992;1:135-142.

Gots RE. Multiple chemical sensitivities: distinguishing between psychogenic and toxicodynamic. *Regul Toxicol Pharmacol.* 1996;24:S8-15.

Gots, R. E., Layton, N. J., & Pirages, S. W. 2003, "Indoor health: background levels of fungi", *AIHA J (Fairfax, Va.)*, vol. 64, no. 4, pp. 427-438.

Gots RE. Medical causation and expert testimony. *Regul Toxicol Pharmacol.* 1986;6:95-102.

Gots RE. Scientific and legal approaches to proof of causation in tort cases. *3rd Chemical Congress of North Am: the book of abstracts ACS.* 1988.

Hertzman C, Hayes M, Singer J, Highland J. Upper Ottawa street landfill site health study. *Environ Health Perspect.* 1987;75:173-195.

Hayes M, Singer J, Highland J. Upper Ottawa street landfill site health study. *Environ Health Perspect.* 1987;75:173-195.

Hopwood DG, Guidotti TL. Recall bias in exposed subjects following a toxic exposure incident. *Arch Environ Health.* 1988;43:234-237.

Hurras, J et al, "Medical Diagnostics for Indoor Mold Exposures" The German Society of Hygiene, Environmental Medicine and Preventive Medicine, *Intl J Hygiene and Environmental Health*, 220 (2017), 305-328.

Institute of Medicine (U.S.). Committee on Damp Indoor Spaces and Health. 2004, *Damp indoor spaces and health.* National Academies Press, Washington, DC.

Jezak K, Kozajda A, Sowiak M, Brzeznicki S, Bonczarowski M, Szadkowska-Stanczyk I, "The Capability of Fungi Isolated from Moldy Dwellings to Produce Toxins." *Intl Journ. of Occ. Med. and Environmtl Health.* 2016, vol. 29, no.5, pp 823-836.

Kabat GC. Introduction: Toward a sociology of health hazards in daily life. In: Kabat GC, ed. *Hyping health risks: Environmental hazards in daily life and the science of epidemiology.* New York: Columbia University Press; 2008:1-12.



Kaye WE, Hall HI, Lybarger JA. Recall bias in disease status associated with perceived exposure to hazardous substances. *Ann Epidemiol*. 1994;4:393-397.

Kellner R. Somatization. Theories and research. *J Nerv Ment Dis*. 1990;178:150-160.

Kelman, B. J., Robbins, C. A., Swenson, L. J., & Hardin, B. D. 2004, "Risk from inhaled mycotoxins in indoor office and residential environments", *Int J Toxicol*, vol. 23, no. 1, pp. 3-10.

Kuhn DM, Ghannoum MA. Indoor mold, toxigenic fungi, and *Stachybotrys chartarum*: infectious disease perspective. *Clin Microbiol Rev*. 2003;16:144-172.

Last JM, Wallace RB. *Public Health and Preventive Medicine, 13th Edition*. Norwalk, CT: Appleton & Lange; 1992.

Lees-Haley PR, Brown RS. Biases in perception and reporting following a perceived toxic exposure. *Percept Mot Skills*. 1992;75:531-544.

Lipscomb JA, Goldman LR, Satin KP, Smith DF, Vance WA, Neutra RR. A follow-up study of the community near the McColl waste disposal site. *Environ Health Perspect*. 1991;94:15-24.

Lipscomb JA, Satin KP, Neutra RR. Reported symptom prevalence rates from comparison populations in community-based environmental studies. *Arch Environ Health*. 1992;47:263-269.

Logue JN, Fox JM. Residential health study of families living near the Drake Chemical Superfund site in Lock Haven, Pennsylvania. *Arch Environ Health*. 1986;41:222-228.

Stachybotrys chartarum: infectious disease perspective. *Clin Microbiol Rev*. 2003;16:144-172.

Manique R, Pena A, Lino CM, Molto JC, Manes J. Ochratoxin A in the morning and afternoon portions of urine from Coimbra and Valencian populations. *Toxicon*. 2008;51:1281-1287.

McCoy LF, Scholl PF, Schleicher RL, Groopman JD, Powers CD, Pfeiffer CM. Analysis of aflatoxin B1-lysine adduct in serum using isotope-dilution liquid



chromatography/tandem mass spectrometry. *Rapid Commun Mass Spectrom.* 2005;19:2203-2210.

Pena A, Seifrtova M, Lino C, Silveira I, Solich P. Estimation of ochratoxin A in portuguese population: new data on the occurrence in human urine by high performance liquid chromatography with fluorescence detection. *Food Chem Toxicol.* 2006;44:1449-1454.

Pennebaker JW, Epstein D. Implicit psychophysiology: effects of common beliefs and idiosyncratic physiological responses on symptom reporting. *J Pers.* 1983;51:468-496.

Pennebaker JW. Psychological bases of symptom reporting: perceptual and emotional aspects of chemical sensitivity. *Toxicol Ind Health.* 1994;10:497-511.

Roht LH, Vernon SW, Weir FW, Pier SM, Sullivan P, Reed LJ. Community exposure to hazardous waste disposal sites: assessing reporting bias. *Am J Epidemiol.* 1985;122:418-433.

Rao CY, Riggs MA, Chew GL et al. Characterization of airborne molds, endotoxins, and glucans in homes in New Orleans after Hurricanes Katrina and Rita. *Appl Environ Microbiol.* 2007;73:1630-1634.

Segerstrom, SC, Miller, GE. Psychological Stress and the Human Immune System: A Meta-Analytic Study of 30 Years of Inquiry. *Psychol Bull.* 2004; 130(4); 601-630.

Thornton, LM, Anderson, BL. Psychoimmunology Examined: The Role of Subjective Stress. *Cellscience.* 2006, 2(4) 1-18.

Shoemaker RC, House DE. A time-series study of sick building syndrome: chronic, biotoxin-associated illness from exposure to water-damaged buildings. *Neurotoxicol Teratol.* 2005;27:29-46.

Shoemaker RC, House DE. Sick building syndrome (SBS) and exposure to water-damaged buildings: Time series study, clinical trial and mechanisms. *Neurotoxicol Teratol.* 2006;28:573-588.



Shoemaker, RC, House, D., and Ryan, JC, Structural Brain Abnormalities in Patients with Infammatory Illness Acquired Following Exposure to Water-Damaged Buildings: A Volumetric MRI Study Using NeurQuant., *Neuroxicolo and Teratol.* 2014; 18-26.

State of California, Health and Human Services Agency, and Department of Health Services. Immunosciences Laboratory, Inc. deficiencies RE: CLIA. Vojdani, A. 1-35. 1-6-2006. San Francisco,CA, San Francisco Regional Office; California Department of Health and Human Services.

Sudakin D, Fallah P. Toxigenic fungi and mycotoxins in outdoor, recreational environments. *Clin Toxicol (Phila)*. 2008;1-7.

Vatinno R, Aresta A, Zambonin CG, Palmisano F. Determination of ochratoxin A in human urine by solid-phase microextraction coupled with liquid chromatography-fluorescence detection. *J Pharm Biomed Anal.* 2007;44:1014-1018.

World Health Organization Guidelines for Indoor Air: Dampness and Mould. 2009 World Health Organization. Copenhagen, Denmark.

